EVALUATION OF FLUID THERAPHY IN INDUCED PYLORIC OBSTRUCTION IN CATTLE

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IN
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BY

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Dedicated to

My Adorable Father for the Silent

Benediction from his Prolonged Sick-Bed

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Dated the 22nd April,1991

C E R T I F I C A T E

This is to certify that the thesis entitled "EVALUATION OF FLUID THERAPY IN INDUCED PYLORIC OBSTRUCTION IN CATTLE" submitted for the degree of Master of Veterinary Science in the subject of Surgery of the Orissa University of Agriculture and Technology, Bhubaneswar is a faithful record of bonafide and original research work carried out by Suresh Chandra Pradhan under my guidance and supervision and that no part of the thesis has been submitted for any other degree or diploma.

The assistance and help received during the course of investigation have been fully acknowledged.

Major Advisor

C E R T I F I C A T E

This is to certify that this thesis entitled "EVALUATION OF FLUID THERAPY IN INDUCED PYLORIC OBSTRUCTION IN CATTLE" submitted by Suresh Chandra Pradhan to the Orissa University of Agriculture and Technology, Bhubaneswar in partial fulfilment of the requirements for the degree of Master of Veterinary Science in the subject of Surgery has been approved by the student's Advisory Committee after an oral examination on the same in collaboration with an External Examiner.

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Suresh Chandra Pradhan

(Suresh Chandra Pradhan)

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CHAPTER I

INTRODUCTION

INTRODUCTION

The disorders of the gastro intestinal tract in ruminants amounts to colossal economic loss either through decreased production or to the life of the animal. Obstructive disorders of the gastro intestinal tract due to different reasons have commonly been reported. The losses encountered have been mainly due to imbalance brought about in fluid and electrolytic status. This is especially true in high intestinal obstruction. Correction of serious imbalances in fluid and electrolytes has been considered to be life sustaining and specially so during the time required for diagnosis and employment of specific therapy.

Pyloric obstruction in cattle is a serious melady and most often the cases are reported only during autopsy. Unlike the single stomached animals, ruminants will have internal vomition leading to sequestration of fluid in the forestomach and serious fluid and electrolyte imbalances. The pathophysiological changes due to pyloric obstruction are so alarming and fast in progression that it will be difficult to take up the specific therapy, unless the patient is brought to sustainable condition. Although it is possible to predict the type of imbalance in the fluid and electrolytes in the obstructive disorders of gastrointestinal tract through history and clinical signs, yet it is difficult to estimate the magnitude of changes. This is mainly due to the contineous alterations in the fluid and electrolyte balance during the course of the disease. As such, reassessment of the clinical and laboratory findings do help to predict in providing fluids having different composition and electrolytes so as to counteract the serious imbalance in fluid and electrolytes.

Perusal of the literature relating to high intestinal obstruction in general and pyloric obstruction in particular revealed to have abnormal changes in fluid and electrolytes. Basing on these changes, specific fluid or the composition of electrolytes in fluid to be infused to the patient suffering from pyloric obstruction, has not yet been systematically worked out. Taking this aspect into consideration, an experimental study has been undertaken to evaluate the clinical signs, biochemical and electrolyte changes. Simultaneously, three different fluids having different concentrations of electrolytes and dextrose have been put into test to assess the eficacy through improved fluid and electrolyte balance and longivity. This will provide a clue to the field veterinarian for appropriate management of the bovine patient suffering from pyloric obstruction.

* * *

CHAPTER II

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Incidence and etiology.

The obstruction of pylorus due to accumulation of sand in cattle was first recorded by Chinayya (1930). Though Wooldrige (1934) has opined about the possibility of occurrence of complete or partial pyloric obstruction, it was Hoflund (1940) who demostrated the effect of sectioning various branches of vagus on the functional impairment of fore-stomach and stenosis of pylorus. Subsequently, workers have reported the incidence of pyloric obstruction due to various reasons such as sand, Gravel (Moller-Sorensen,1960; Pinsent,1962; Esperson,1964 and Hunter 1975), hair balls, foreign bodies (Pope, 1961; Fox, 1965; Merritt and Boucher, 1967; Whitlock,1980 and Blood et al., 1985), Coarse food material (Wyssmann, 1929 and Ashcroft, 1983) and infected fodder (Esperson 1964 and Mathison et al., 1981). There are several other conditions like advanced stage of pregancy (Hutchins et al.,1957; Merritt and Boucher, 1967 and Blood et al., 1985), compression by tumours (Biscoff,1953 and Blood et al.,1983), presence of ulcers (Esperson, 1964), abomasal catarrh

(Moller-Sorenson 1960), traumatic reticulitis (Begg,1950 and Blood et al.,1983), injury to vagus (Naerland and Helle,1962; Neal and Edwards, 1968 and Blood et al., 1985), dilatation and displacement of abomasum (Neal and Pinsent 1960, Corker and Dziuk, 1968; Whitlock, 1969 and Blood et al.,1985) and as a consequence to secondary indigestion (Kuiper and Breukink, 1986 b) may subject the animals to suffer from pyloric obstruction. A 5 day old cross-bred cow calf have been reported by Singh et al.(1989) to have also been affected with pyloric obstruction.

Symptoms:

The most significant physical symptoms in both affected and experimental cases of pyloric stenosis manifested by anorxia, dehydration, depression, muscular weakness, apathetic and indifference to normal stimuli, dropping of ears, shruken eyes, dry muzzle, distended abdomen cold extremities and loss of appetite were observed by many workers (Pope,1961; Naerland and Helle,1962; Pinsent,1962; Esperson,1964; Merritt and Boucher,1967; Corker and Dziuk,1968; Ashcroft,1983; papadopoulos et al.,1985 a; Kuiper and Breukink,1986 a; Mishra,1987; Basu,1987; Singh et al.,1989; Braun et al.,1990 and Sethy, 1990). Contrary to these opinions Kuiper and Breukink (1986 a) have opined that it is difficult to distinguish between affection of fore-stomachs and pyloric obstruction through symptoms alone.

An enlargement of abdomen in 'L' shape from left to right when viewed from behind the animals has been observed by Jones and Pirie (1962), Naerland and Helle (1962), Pinsent (1962), Neal and Edwards (1968), Whitlock (1969), Dass et al.(1981a),Fubini et al.(1985) and Kuiper and Breukink (1986 a).

Pulse:

Hoflund (1940) and Hutchins et al.(1957) have recorded an increase in pulse rate in pyloric obstruction. Similar observation was also made by Whitlock (1969), Ferrante and Whitlock (1981), Asheroft (1983), Pathy (1986), Mishra (1987), Basu (1987) and Sethy (1990) in affected and experimental cases.

Respiration rate:

Esperson (1964) has observed respiration rate to be in the normal range in animals suffering from pyloric obstruction. Identical observation was made by Pathy (1986), Mishra(1987) and Sethy (1990) in experimentaly induced pyloric obstruction in calves. However, Papadopoulos (1985 a) recorded progressively shallow respiration with expiratory grunt while Basu et al.(1990) reported a significant increase in respiration of animals in experimentally induced pyloric obstruction.

Rectal temperature:

There are contradictory opinions regarding the changes in rectal temperature of animals affected with pyloric obstruction. Hutchins et al. (1957), Jones and Pirie (1962), Pinsent (1962), and Ferrante and Whitlock (1981) reported it to be in normal range, while Ashcroft (1983) and Papadopoulos et al. (1985 a) recorded it to be either normal or low. Mishra (1987) has stated that body temperature fluctuated within the first 72 hours of obstruction and thereafter reached subnormal level till death. Basu et al. (1990) have recorded reduced rectal temperature in experimentally induced pyloric obstruction in calves.

Faeces and Urine:

Pinsent (1962) has stated that scanty faeces was voided by the animal in pyloric obstruction which was also confirmed by Neal and Edwards (1968) and Dass et al.(1981 b). On the other hand Papadopoulos et al.(1985 a) have reported that scanty faeces was passed by the animals in this condition for 1 to 2 days followed by complete stoppage. The faeces, in their opinion, was usually covered with mucous and there was also significant decrease in the volume of urine. Similar observations were also made by Basu (1987), Mishra (1987) and Braun et al.(1990).

Dehydration:

The onset of dehydration according to Pope (1961), Robertson (1966), Gingerich and Murdick (1975), Ferrante and Whitlock (1981), Asheroft (1983), Papadopoulos et al.(1985 a), Kuiper and Breukink (1986 b), Basu (1987), Mishra (1987) and Braun et al.(1990) was was reported to be the most important and common symptom during pyloric obstruction. The dehydration of animals progresses rapidly with progression of the disease. Varying degrees of dehydration has been noticed in intestinal obstruction in bovine by Rathor et al. (1977).

Appetite:

The appetite was recorded to be decreased by Hutchins et al.(1957), Pinsent (1962), Esperson (1964), Fox (1965), Corker and Dziuk (1968), Whitlock (1969), Poulsen (1974), Papadopoulos _et al_.(1985 a), Kuiper and Breukink (1986 b), Basu (1987) and Mishra (1987) in cattle in both affected and induced pyloric obstruction.

Rumen Motility:

Ruminal motility was reported to be absent during pyloric obstruction by Hutchins _et al_.(1957) whereas, Pope (1961) has observed it to be less than 1 in every 3 minutes and weaker than normal. Suspended rumination in identical cases was observed by Pinsent (1962) and Dass _et al_. (1981 b) which was also reported by Rathor _et al_. (1977) and Singh _et al_. (1985) in cases of intestinal obstruction in cattle. The study in experimental cases by Basu (1987) and Mishra (1987) did reveal to have decreased ruminal contraction which stopped after 72 hours. The atony of the rumen from the first day was observed by Papadopoulos _et al_. (1985 a) with virtually no contraction after second day. They have also recorded that rumination stopped completely from the first day in experimentally produced duodenal and intestinal obstructions. However, Rebhun (1980) has observed that motility and contractibility of rumen was variable. The rate of contraction in his opinion though increased yet the strength of contraction and motility were found to be decreased.

Abdominal sound:

It has been reported by Begg (1950) that a tympanitic viscus could be palpated by him at the border of last rib on right side during pyloric obstruction. They have also recorded high resonance on percussion of this area. Pinging and splashing sounds on ascultation and percussion at right paralumbar fossa in cases of pyloric obstruction was recorded by Whitlock (1969), Dass et al. (1981 a), Ferrante and Whitlock (1981), Pathy (1986), Basu (1987), Mishra (1987) and Sethy (1990).

Muscular Weakness:

Basu et al. (1990) have reported severe muscular weakness as the disease progressed in experimental pyloric obstruction in cattle. Identical observation in cases of duodenal obstruction has been reported by Papadopoulos et al. (1985 a).

Artificially Induced Pyloric obstruction:

In an attempt to induce pyloric obstruction, Hammond et al. (1964) did it by inflating rubber balloon which was passed through a canula or a fistula. Subsequently, they preferred to ligate the pylorus with a venyl plastic tubing ligature through which a wooven cotton thread was passed to bear the tension. Corker and Diuik (1968) modified the method of Hammond et al. (1964). They ligated the pylorous and passed the ligature through two plastic discs placed one inside and the other outside the abdoninal wall before tightening. This they claimed helped in preventing the adhesion.

Papadopoulos et al.(1985 a) induced pyloric obstruction in cows by passing a flexible plastic tubing around the bowel in double loop fashion with two plastic discs fixed to the abdominal wall to prevent adhesion. Purohit et al. (1984 a) created obstruction to the duodenum of sheep by applying polythene tube threaded with cotton lace which was passed through the holes of buttons placed against the duodenal wall and the external surface of the skin. Subsequently, Basu et al. (1990) ligated the pylorus directly with umbilical tape avoiding the blood vessels. The site of approach to pylorus was made through right paralumbar fossa behind the last rib by Biscoff(1953) whereas, Lowe et al.(1961), suggested a paramudian approach for pylorus. Pathy (1986), Basu (1987) and Sethy (1990) have claimed that right lower paracastal approach was the best to approach the pylorus in cattle.

Packed Cell volume (PCV)

An increase in PCV has been recorded by different workers in cases of pyloric obstruction in experimental cases (Basu et al.,1990) which was also the observation in 3 out of 10 cows by Braun et al. (1990) while the rest 7 had normal PCV level.

It has been reported that an increase in PCV was also observed in duodenal obstruction (Hammond et al.,1964; Papadopoulos et al., 1985 b and Avery et al., 1986), right side abomasal displacement (Poulsen, 1974 and Smith,1978), intestinal infarction (Chandrababu and Ramakrishna, 1981) abomasal impaction (Ashcroft,1983), and intestinal obstruction (Purohit et al.,1984 a and Smith, 1985 a and 1985 b).

Haemoglobin:

Kuiper and Breukink (1986 a) have reported to have observed an increase in haemoglobin level in cows which was also recorded by Basu et al.(1990) in experimentally induced pyloric obstruction in bovine.

Jones (1952) and Dass et al.(1981 a) did observe significant fall in haemoglobin in animals with abomasal displacement, but Poulsen (1974) has recorded a rise in the level. Higher haemoglobin values were also recorded by McGuirk and Butler (1980) in case of metabolic alkalosis in cattle. Purohit et al. (1984 a) have observed significant increase in haemoglobin during intestinal obstruction in sheep which was recorded by Papadopoulos et al. (1985 b) in induced cases in bovine. Kuiper and Breukink (1986 a) too recorded an increase in haemoglobin level in cases of recticulo-omasal stenosis.

Biochemical changes in blood and serum:

Glucose:

Hutchins et al. (1957) have recorded low blood glucose level in two bovine suffering from pyloric stenosis with ketotic symptoms. Pathy (1986) has found hypoglycaemia in early stages of pyloric obstruction in experimental animals which increased significantly at the terminal stages. Basu et al. (1990) while supporting (Pathy 1986) have stated that hypoglycaemia at the terminal stages was due to release of glucose to compensate the severe stress.

Pope (1961) have observed a significant increase in blood glucose level in the terminal stages of abomasal impaction, but Singh and Kohli (1985) have recorded an initial hyperglycaemia followed by significant hypoglycaemia at later stages of jejunal obstruction in experimental calves. A mild increase in serum glucose level of experimental animals with duodenal obstruction was recorded by Avery et al. (1986). Low blood glucose level have been recorded in intestinal infarction by Chandrababu and Ramakrishna (1981).

Calcium:

The normal serum calcium level in healthy bovine has been studied by many workers and it was found to vary between 9 to 12 mg/dl (Bhatia et al., 1972; Malik et al., 1974; Pandey et al., 1980 and Pyne and Maitra, 1982).

Kuiper and Breukink (1986 b) carried out detailed investigation in 23 cows suffering from secondary indigestion leading to functional pyloric stenosis and have recorded a decrease in blood calcium level. On the contrary, it was reported by Ellis (1987) that the level was not reduced in human being in identical condition. Pathy (1986) has recorded a significant fall in serum calcium level following experimental ligation of pylorus in calves. Basu et al. (1990) did observe mild hypocalcaemia in experimental calves with pyloric obstruction.

Purohit et al. (1984 b) have observed significant variation in calcium level after both high and low intestinal obstruction in sheep. This level was found to be not changed in experimental heifer with duodenal obstruction.

Potassium

The normal plasma potassium in cattle was recorded to be 4.7 ± 0.48 MEq/L. by Mylera and Bayfield (1968) which they have opined decreases slightly with age,

Hypochloraemic hypokalaemic metabolic alkalosis is also observed in pyloric stenosis in bovine (Kuiper and Breukink, 1986 b; Basu et al., 1990 and Braun et al., 1990).

The condition of hypokalaemic hypochloraemic metabolic alkalosis was reported to occur in abomasal disorders too in bovine as has been established by Gingerich and Murdick (1975), Dass et al.(1981 a and 1981 b), Whitlock (1980), Lattman (1984) and Purohit et al.(1984 b). The same observation was made by Papadopoulos et al.(1985 b) and Smith (1985 b) in intestinal obstruction, caecal dilatation and volvulus in cattle.

Hammond et al.(1964) and Avery et al.(1986) have recorded significant hypokalaemia in duodenal obstruction in bovine. In their opinion, hypokalaemia was probably due to reduced feed intake and movement of potassium into cell as a result of alkalosis. Campbell et al.(1986) have recorded hypokalaemic condition in gastro-intestinal ulcer disease in 14 foals.

Sodium:

The normal sodium level was estimated to be 141 ± 5.2 mEq./L. in bovines by Mylers and Bayfield (1968).

Robertson(1965 and 1966) and Smith (1978) recorded hyponatremia in abomasal displacement and torsion and stated it to be due to diarrhoea and dehydration. However, Gingerich and Murdick (1975), Das et al. (1981 a and 1981 b) and Ashcroft (1983) did not find any significant change in serum sodium level in similar cases. Ashcroft (1983) also suggested that hypochloraemia with normal sodium concentrations was the indication of failure of ingesta transport from abomasum to small intestine.
Purohit et al. (1984 b) recorded significant fall in serum sodium level in high intestinal obstruction in sheep.

Papadopoulos et al.(1985 b) did not record any essential change of sodium in intestinal and doudenal obstruction in bovine while Avery (1986) reported slight decrease after 72 hours of duodenal obstruction in bovine.

Hyponatremia was recorded in human being during duodenal obstruction by Ellis (1987).

In the opinion of Pathy (1986) and Basu et al.(1990) Sodium level did not show any deviation in induced pyloric obstruction in calves.

Chloride:

Hammond et al.(1964) and Avery et al.(1986) have recorded hypochloraemic condition in duodenal obstruction. In their opinion, this condition was due to reflux of abomasal contents into the rumen causing increased ruminal fluid chloride concentration, Whereas, Kuiper and Breukink(1986 b), Braun et al. (1990) and Basu et al.(1990) did find hypochloraemia in pyloric obstruction. Identical change in chloride level was recorded by Ellis (1987) in his study in pyloric stenosis in human being. Hypochloraemia has been reported by Robertson (1965 and 1966), Gingerich and Murdick(1975), Das et al. (1981 a, 1981 b), Ashcroft (1983), Rings et al.(1984) and Lattman (1984) to be common occurrence in abomasal displacement, torsion or impaction. Similar observation was also recorded by Purohit et al. (1984 b), Papadopoulos et al. (1985 b), Smith (1985 a), Avery et al. (1986) and Pearson (1971) in intestinal obstruction, too.

Blood Urea nitrogen (BUN)

Campbell (1970) has stated that elevated BUN levels indicate an upset in fluid and electrolyte status and if it persists, then it indicates the grave condition of the disease.

Hammond et al. (1964) recorded an elevated BUN levels in calves with intestinal obstruction, while Krishnamurty et al.(1980) have observed the same in buffalo calves. The same observations were made by Purohit et al.(1984 b), Papadopoulos et al.(1985 b) in cattle suffering from intestinal obstruction.

Ashcroft (1983) has studied 27 cases of abomasal impaction in cattle in which he observed elevated uremia upto 26.2 m Mol/L. Singh et al.(1985) have observed increase in BUN in case of dilatation and torsion of caecum in a cow.

Kuiper and Breukink (1985 b) recorded some degree of uremia in pyloric stenosis in cows which Ellis (1987) reported in pyloric stenosis in human being.
Avery et al.(1986) have stated that the serum concentration of urea nitrogen was found to be increased in duodenal obstruction in heifers. In his opinion, the increase in urea nitrogen value in the blood is due to dehydration

and prerenal azotaemia associated with hypochloremic alkalosis. Braun et al.(1990) and Sethy (1990) have observed significant increase in BUN in pyloric stenosis in bovine.

Creatinine:

The normal creatinine value in Gir and Jersey cows was estimated by Kulkarni et al.(1984) and it was found to be 1.20 $\pm$ 0.03 and 0.99 $\pm$ 0.02 mg/dl.

Singh et al.(1985) have observed creatinine to be above normal range in case of dilatation and torsion of caecum in a cow.

Avery et al.(1986) reported significant increase in creatine level in experimentally induced duodenal obstruction in bovine. They attributed it to be due to decreased extra cellular volume and prerenal azotaemia associated with hypochloremic alkalosis.

Fluid Therapy:

Clinical efficacy of common fluids used to correct various body disorders have been analysed by many workers.

Kaneko (1971) was of the opinion that the rational fluid should have such composition that which when administered will complement the ECF of the patient.

Different fluids commonly used by different workers have been summerised.

1. <u>Solutions of Dextrose:</u>

The administration of dextrose solutions causes decrease in the concentration of electrolytes as the dextrose is metabolised according to Kaneko and Cornelius(1971) and Coldston <u>et al</u>.(1983).

Michel (1977), Clark (1980) and Michel (1983) have stated that 5 per cent dextrose solution is isotonic and provides energy of 790 KCL/L and replaces the body water in cattle, whereas, hypertonic solutions of dextrose 20 per cent or 50 per cent have been suggested to be used in the body conditions associated with hypoglycaemia (Coldston <u>et al</u>.,1983 and Michel, 1983). However, while agreeing with the earlier workers Blood <u>et al</u>.(1985) have also stated that dextrose does promote the movement of ECF potassium into the cell.

2. <u>Saline Solutions:</u>

These solutions contain water and sodium chloride. Isotonic saline or normal saline solution containing 0.9 per cent sodium chloride has sodium and chloride ioms and water. Although it has the same osmolarity as body

ECF regarding sodium ion concentration, but has got high concentration of chloride ions. When given alone it simply ehydrate the patient without replacement of other electrolytes of plasma and will agravate hypokalaemia or metabolic acidosis (Kaneko and Cornelius, 1971). It is intended to expand the blood volume in case of hypovolemic shock and dehydration according to Blood et al.(1985).

Reaves _et al_. (1983) have stated that 5 per cent saline may be used for dialysis whereas, Michel(1983) did not find any beneficial effect by using hypertonic saline solutions in animals. Hypertonic solution of sodium chloride if injected causes reduction of flow of water into the cell causing its retention in ECF.

The normal saline solution in the opinion of Clark (1980) is suitable for the replacement therapy. It's major use is in the treatment of gastric vomition where acid is lost leading to alkalosis. It is also mostly indicated incasesin pyloric obstruction in bovine.

Coldston (1983) has advocated that Hypotonic saline (0.4 per cent Nacl) is useful for treating hypernatremia and replacing deficit for water.

3. <u>Ringer's Solution:</u>

Kaneko and Cornelius (1971) and Coldston *et al.*(1983) have stated that Ringer's solution is a balanced electrolyte containing sodium, potassium, chloride and calcium ions. The solution causes rehydration without aggravating or inducing electroneutrality disturbances. It was stated by them to be useful in metabolic alkalosis brought on by profound vomition.

4. <u>Lactated Ringer's solution/Hartmann solution.</u>

Clark (1980), Coldston (1983), Blood *et al.*(1985) and Michel (1983) have stated that Lactated Ringer's solution containing physiological concentration of sodium Potassium, Calcium and chloride together with 28 mEq/L. lactate can be used in acidosis diarrhoea, gastromiterstinal vomiting and dehydration. The lactate is metabolised into bicarbonate ion by the liver.

Esperson (1964) has recommended sodium chloride, glucose, lactic acid, potassium chloride, ammonium chloride in cases of dehydration and alkalosis in cattle.

Kaneko and Cornelius (1971) and Coldston (1983) also advised 1/6th molar lactate or 1.3 per cent sodium bicarbonate for treatment of metabolic acidosis. This solution should be given very carefully so as not to overload the patient with sodium.

Gingerich (1975) has stated that the infusion of balanced electrolyte solutions rich in chloride such as Ringer's solution might be life saving in seriously debilitated cattle due to abomasal displacement or disorders when sequestration of chloride occurs in the abomasum or fore-stomach leading to hypochloraemia.

Rathor _et al_.(1977) did administer glucosaline and Ringer's lactate solutions two days prior to surgery in three cows suffering from intestinal obstruction.

Aturson (1983) has stated that in the initial phase of rehydration the balanced salt solution must contain 2.5 to 5 per cent glucose to provide energy requirement. When higher amount of glucose is added to infusants it may provoke glycosuria with renal fluid loss and tendency to increase metabolic acidosis due to lactate production.

Singh _et al_. (1985) administered Ringer's solution in case of dilatation and torsion of caecum prior to surgery which gave uneventful recovery. Smith (1985 b) has stated that if the animal is dehydrated preoperative rehydration with I.V. fluid is strongly recommended. He further stated that isotonic saline with potassium chloride added at 1 to 2 gms/L is the recommended type of I.V. fluid supplemented with 5 per cent dextrose. Compbell-Thompson (1986) gave 0.9 per cent sodium chloride I.V. before conducting surgery of gastrointestinal ulcer in 14 foals to check dehydration and volume loss.

Ellis (1987) has stated that when the patient is dehydrated due to pyloric stenosis, there would be electrolyte depletion. He recommended I.V. replacement therapy with normal saline together with potassium supplementation in human being.

Blood et al.(1985) computed some fluids for treatment of specific disease conditions and summarised as follows.

i) Lactated Ringer's solution plus 1 gm/L. potassium chloride and 5 gm/L. sodium bicarbonate is indicated for acidosis, hypernatremia and hypokalaemia.

ii) Isotonic Potassium Chloride 1.1 per cent + Isotonic saline (0.9 per cent) and dextrose 5 per cent is indicated for metabolic alkalosis in cattle with abomasal disorders.

iii) Isotonic Potassium Chloride and ammonium chloride are recommended for treatment for alkalosis associated with right side displacement of abomasum.

iv) Sodium Bicarbonate solutions, 1/6th molar sodium lactate solution or 1.3 per cent sodium bicarbonate isotonic solution are strongly alkalizing solution which are used in case of metabolic acidosis.

v) Sodium bicarbonate 1.3 per cent in 5 per cent dextrose and 5 per cent sodium bicarbonate is used for treatment of mild to severe acidosis.

vi) A mixture of isotonic saline and isotonic sodium bicarbonate can be used for treatment of acidosis.

Dose and rate of Administration:

Dose of each fluid is calculated depending upon the degree of dehydration present in specific disease conditions. Total volume is assessed to replace the volume deficit and then fulfil the maintenance requirement.

Michel (1974) had stated that an animal having body weight loss exceeding 5 per cent due to dehydration probably lost about 50 ml of fluid per kg body weight.

Wingfield et al.(1975) suggested I.V. injection of Ringer's solution at the rate of 30 ml/kg body weight to prevent shock and improve in tissue perfusion.

Clark (1980) while supporting the views of Michel (1974) recommended that the rate of crystalloid infusion should not exceed 20 ml/kg for a maximum of 4 hours.

Michel (1983) has recommended to give half the estimated deficit in 6 hours, three quarters in 24 hours and all within 48 hours. The rate of administration is established to be 8 to 25 ml/kg per hour. Potassium containing solutions are infused at 25 ml/kg per hour and heart rate is monitored carefully during administration.

Felor and Felor (1985) has stated that body weight loss due to fluid loss can be estimated by multiplying percentage of dehydration with total body weight.

Srinivasan (1983) has reported that a solution containing sodium chloride 8.6 gm, potassium chloride 0.3 gm, hydrated calcium chloride 0.48 gm in 1000 ml of distilled water is very helpful to prevent shock and dehydration.

Pathy (1986) did use the solution suggested by Srinivasan (1983) in calves with experimental pyloric obstruction at dose rate of 30 ml/kg body weight. He has claimed to be successful in resuscitating and increasing the longivity of the animals to 153.3 hours as against 94.8 hours without fluid therapy.

Blood et al. (1983) provided the following guide line for replacement of the volume deficit depending on the degrees of dehydration and basing on the skin fold test alongwith body weight loss.

Body weight loss	Shruken eye shruken face	Skin fold test persisting for seconds	Fluid required to replace volume deficit ml/kg BW.
per cent			
4-6	Barely detectable	-	20-25
6-8	++	2-4	20-30
8-10	++	6-10	50-80
10-12	++++	20-45	80-120

They have further stated that fluid is administered in two stages; hydration therapy in the first 4 to 6 hours and maintenance therapy in the next 20 to 24 hours depending on the seriousness of the disease condition and rate of administration of isotonic solution is 3 to 5 litres per hour.

* * *

CHAPTER III

MATERIALS AND METHODS

M A T E R I A L S   A N D   M E T H O D S

A total number of nine clinically healthy cross bred Jersey male calves in the age group of $1\frac{1}{2}$ to 2 years formed the domain of the present experiment. The body weight of these animals varied from 50 to 70 kgs. The animals were dewormed and were maintained in balance feeding and management care for 7 days. The calves were placed in 3 groups with 3 in each, irrespective of age and body weight.

<u>Group-I</u>

The rates of pulse, heart and respiration, rectal temperature and ruminal contraction rate were recorded. Blood samples were collected from the jugular vein with and without anticoagulant from each animal 24 hours before experiment.

Two millilitres of blood was collected in a dry test tube containing about 4 mg of ethylenediamine tetra acctate (EDTA) for haematological studies and blood glucose estimation. A further 10 ml of blood was collected without anticoagulant in a test tube to separate the serum and to estimate calcium, potassium, sodium, chloride, urea nitrogen and creatinine.

Induction of pyloric abstruction

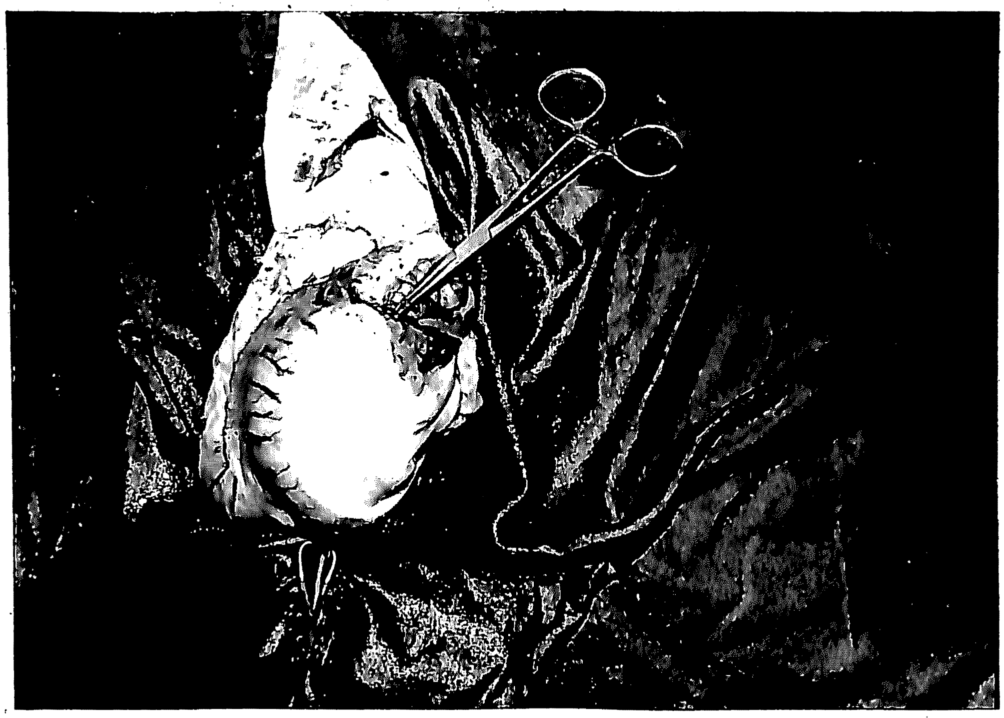
The animals were kept on fasting for 24 hours. They were then restrained in left lateral recumbency. They were prepared for right lower paracostal laparotomy observing conventional surgical toilet.

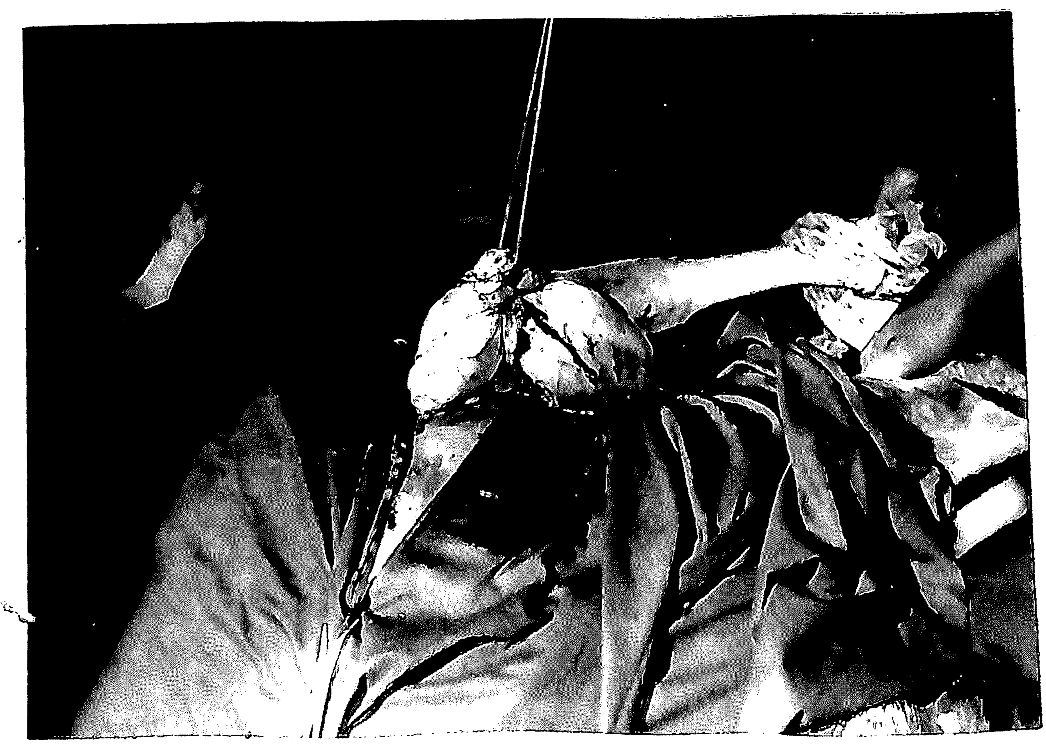
The animals were administered with Triflupromazine hydrochloride (marketed as 'siquil' by Sarabhai chemicals, India Ltd.) intravenously at the dose rate of 1 mg/kg. body weight. The site of incision was selected at a distance of 1.5 cm posterior and parallel to the caudal border of the last rib in the lower paracoastal area. Linear infiltration with 30 ml of 2 percent lignocaine with adrenaline (Marketed by Warren Pharamaceuticals Ltd., India as 'Lignox') was made in two tiers below the skin and within the muscle along the line of incision.

An incision of 10 cm in length was given extending from the lower border of right paralumbar fossa upto the costochondral junction to open the abdomen. The pylorus which was lying ventromedial to the 11th rib was located and exteriorised (Plate1). The omental attachment at the pylorus was separated and blood vessels at the pylorus were identified. A piece of sterilized umbilical tape threaded in a curved atraumatic needle was passed around the pylorus.

Plate 1: Showing the exteriorised pylorus

Plate II: Showing the umbilical tape passed around the pylorus





avoiding the blood vessels (Plate-II). The knot was placed to completely occlude the lumen of the pylorus. It was confirmed by milking the abomasal contents from both the directions. Nitrofurazone solution (Furacin-Vet marketed by Eskayff Ltd., India) was instilled on the ligated part of the pylorus. The abdomen was closed in routine manner. A protective pad soaked with spirit acriflavin was fixed over the suture line (Plate-III). Daily dressing of the would was carried out using spirit acriflavin.

The animals were kept on observation till cessation of defecation developed. It was evident mostly after 72 hours of induction alongwith associated changes indicating pyloric obstruction.

Group-I

The animals of this group was intravenously administered with fluid I. It was prepared in the laboratory and was autoclaved. The fluid contained Sodium chloride 0.9 per cent, Potassium chloride 1.1 per cent, calcium chloride 0.1 per cent and dextrose 5 per cent. The dose rate of infusion was maintained at 30 ml/kg body weight (Wingfield, 1975). The flow rate was monitroed at 25 ml/kg/hour (Michel, 1983). The fluid was adminis-

Plate III: Showing the operated animal with protective pad at the right lower paracastal area.

30 (a)

tered at 24 hours interval till hour post induction. Blood samples were collected at 24 hours interval and the same parameters were investigated which were done before induction.

Group II:

The animals of this group were anaesthetised and induced with pyloric obstruction in identical procedures as was done in Group I. The animals showed signs of obstruction after 72 hours of induction following which Fluid II was initiated. It was also prepared in the laboratory and was autoclaved. The fluid had same composition as in Fluid-I but dextrose 10% was incorporated instead of 5 %. The dose and flow rates were maintained as in Group I. The administration of fluid was done at time interval similar to Group I. The blood samples were obtained and estimated alike group I.

Group-III:

The methods of anaesthesia and creation of pyloric obstruction in calves was followed as was done in the animals of Group I and II. The animals exhibited similar symptoms of obstruction as was observed in Group I and Group II after 72 hours experiment.

The calves were then subjected to intravenous fluid infusion in the same dose and flow rates as was done in

Group I and II. However, the fluid which was used in this group of animals was of the following ingredients and concentrations. They were Sodium Chloride 0.6 per cent, Potassium chloride 0.04 per cent, Calcium chloride 0.027 per cent, Sodium lactate 0.312 per cent and dextrose 20 per cent. It is a commercially marketed fluid 'Rintose' manufactured by Wockhardt Veterinary Limited, India.

Haematological Examinations:

Packed Cell Volume (PCV)

The packed cell volume was estimated by Wintrobe haematocrit tubes and Zatenzki Centrifuse (Sastry, 1972).

Haemoglobin

The whole blood haemoglobin was recorded by using Sahli's haemoglobinometer according to Sastry (1976).

Biochemical Estimation of Blood Glucose, Urea nitrogen and creatinine

Blood glucose, urea nitrogen and creatinine were estimated by spectrophotometric method as described in the instruction manual supplied with spectronic-20 (1965).

Chloride:

Serum Chloride was estimated by the titrimetric method as suggested by Schales and schales (Wootton, 1964).

Calcium:

The serum calcium level was estimated by the Clark and Collip (1925) modification of Kramer-Tisdal method.

Sodium and Potassium:

Sodium and Potassium content of the serum were determined by flame photometry method according to Wootton (1964) using EEL flame photometer.

The data were analysed by analysis of variance as has been suggested by Snedecor and Cochran (1967) and are tabulated in Table 2, 3, 4, 5 and 6.

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CHAPTER IV

RESULTS

R E S U L T S

Pyloric obstruction was induced in nine experimental animals and were observed for changes in different visible symptoms. Clear symptoms of obstruction were recorded at 72 hours as the animals stopped passing of faeces. Fluid therapy was given at 72 hours post ligation and was repeated at an interval of 24 hours till the death of the animals or 192 hours post ligation. The changes in different parameters were recorded till 192 hours in the surviving calves.

The site of operation was observed to have developed postoperative inflammatory swelling which disappeared by 72 hours after induction. The healing of the wound was uneventful. The animals were assigned in 3 groups with 3 in each group. Three differently composed fluid was administered intravenously to 3 groups of calves. The mean period of survival of the animals were recorded (Table-I) to be 146.3 and 161 hours in Group-I and II respectively. All the 3 animals survived till 192 hours of investigation in Group-III.

Table I Showing period of survival of the Experimental Calves following ligation of pylorus

Groups	Fluid	Calf	Period of survival in hours	Average of the period or survival
		1	143	
I	1	2	138	146.3
		3	158	
		1	163	
II	II	2	156	161
		3	164	
		1	192	Sacrificed at 192 hours
III	III	2	192	
		3	192	

Group Treatment:

Group I Fluid No. I Containing Potassium Chloride 1.1 gm, Sodium Chloride 0.9 gm, Dextose 5 gm, Calcium Chloride 0.1 gm in 100 ml of distilled water.

Group II Fluid No. II Containing Potassium chloride 1.1 gm, Sodium chloride 0.9 gm, Dextose 10gm, Calcium Chloride 0.1 gm in 100 ml of distilled water.

Group III Fluid No.III Containing Potassium chloride 0.04 gm, Sodium Chloride 0.6gm, Dextose 20 gm, Calcium Chloride 0.027 gm, Sodium lactate 0.312 gm in 100 ml of distilled water.

Plate IV: Showing back view of the operated animal with dorso-ventral distension on left and ventro-lateral distension on right side abdomen at 72 hours post pyloric obstruction.

37 (a)

therapy at 24 hours interval it was observed that the intensity of dehydration did not increase further.

Mucoid discharge:

Thick mucoid nasal discharge was observed in all the animals after 72 hours post-obstruction.

Defecation:

Animals of all the three groups voided scanty, dry and hard pelleted faeces upto 72 hours of pyloric obstruction. Thereafter, the animals were straining and were voiding blood tinged mucous. Intestinal motility was recorded on palpation and ascultation at right lower abdominal area soon after fluid therapy.

Urination:

There was apparent gradual reduction in the volume of urine in all the animals after obstruction. Reduction in urinary volume was noticed which appeared to increase in Group III animals after fluid therapy.

Pulse rates:

The pulse rate of the control period of the animals in all three groups varied between

60.67 ± 0.54 to 65.00 ± 0.94 per/minute (Table 2). The rate showed an increasing trend (Fig. 1) in Group I till 120 hours after obstruction and 48 hours after infusion of Fluid I. The values were significant ($P < 0.05$) from one another (Appendix. 1 a, 2 a, 3 a). Analysis of variance has revealed that the mean values at different intervals following fluid therapy were significantly ($P < 0.01$) increasing than the control level. The animals of Group II following infusion of Fluid II also showed an increase ($P < 0.05$) till 120 hours with highest value of 100.67 ± 0.54 per minute. Thereafter, it dropped significantly ($P < 0.05$) at 144 hours (Fig. I). The mean values at 72 and 96 hours were not significant from each other (Appendix 1 a, 2 a, 3 a). Analysis of variance has revealed that the mean values at different intervals following fluid therapy were significantly ($P < 0.01$) increasing than the control level. The animals of Group II following infusion of Fluid II also showed an increase ($P < 0.05$) till 120 hours with highest value of 100.67 ± 0.54 per minute. Thereafter, it dropped significantly ($P < 0.05$) at 144 hours (Fig. 1). The mean values at 72 and 96 hours were not significant from each other (Appendix 1 a, 2 a, 3 a). The mean values of pulse rate in the animals of group III after administration of Fluid III gradually increased till 192 hours and was recorded to be 102.67 ± 2.33 per minute. The values were variably significant ($P \angle 0.05$) than one another during the entire period of investigation.

Heart beat:

The mean level of heart beat in the control period of animals of all the groups ranged between 67.67 ± 0.54 and 70.33 ± 0.27 per minute (Table 2). The mean values in the animals of group I increased even after fluid therapy and reached the mean of 100.33 ± 0.72 per minute. However, the values at 96 and 120 hours were not significant from each other (Appendix 1 a). The mean values of heart beat in animals of Group II and similar increase ($P \angle 0.05$) till 120 hours (Fig. 2, Table 2). Subsequently, there was a drop in the mean values and was found to be not significant than the control. The changes in mean values at 72, 96 and 120 hours after fluid therapy were not significant from one another. The mean values of heart beat in Group III increased significantly ($P \angle 0.05$) till 168 hours. It subsequently showed a fall at 192 hours. The values from 120 to 192 hours were recorded to be not significant than one

Table 2 Showing changes (Mean ± SE) in Pulse and heart beat, before and after pyloric obstruction and after fluid therapy in experimental calves.

Parameters	Groups	Before obstruction	After obstruction					
			H	O	U	R	S	
		24 =	72 I.V. fluid	96	120	144	168	192
Pulse rate (per minute)	I	60.67±0.54[a]	88.33±0.27[b]	94.00±2.49[c]	99.67±0.72[d]	-	-	-
	II	65.00±0.94[a]	88.33±0.72[b]	91.00±0.82[b]	100.67±0.54[d]	96.00±0,47[c]	-	-
	III	63.33±0.54[a]	72.33±3.21[a]	87.67±5.86[b]	90.00±4.92[bc]	95.33±5.80[bc]	101.33±2.60[bc]	102.67±2.33[c]
Heart beat (per minute)	I	67.67±0.54[a]	90.33±0.72[b]	97.33±1.52[c]	100.33±0.72[c]	-	-	-
	II	70.33±0.27[a]	100.00±2.16[b]	102.00±2.49[b]	105.67±1.52[b]	68.30±12.22[a]	-	-
	III	69.67±0.27[a]	79.67±2.72[b]	86.33±1.36[c]	99.33±2.93[cd]	101.33±0.72[d]	103.67±0.98[d]	102.00±1.25[d]
		(3)	(3)	(3)	(3)	(3)	(3)	(3)

= Control, Figures in parantheses indicate number of animals.

Group treatment: Group I: Fluid containing Potassium chloride 1.1 gm, Sodium Chloride 0.9 gm Dextrose 5 gm, Calcium chloride 0.1 gm in 100 ml of distilled water.

Group II Fluid containing Potassium chloride 1.1 gm, Sodium Chloride 0.9 gm Dextrose 10 gm Calcium chloride 0.1 gm in 100 ml distilled water.

Group III Rintose containing Potassium chloride 0.04 gm, Sodium Chlride 0.6 gm Dextose 20 gm, Calcium Chloride 0.027gm and Sodium lactate 0.312 gm.

Identical superscipts indicate not significant, Different superscrips indicate significant ($P < 0.05$)

another according to analysis of variance (Appendix 1 a, 2 a, 3 a). The mean levels in all the groups were found to be significantly ($P < 0.01$) higher than the control level after introduction of fluid therapy.

Respiration:

The mean respiration rate of the control animals was found to be ranging between 21.33 ± 0.27 and 24.00 ± 0.47 per minute (Table 3). The mean level showed a gradual and significant ($P < 0.05$) decrease till 120 hours in Group I (Fig. 3). The values varied significantly ($P < 0.05$) from one another at all intervals of time after induction of fluid therapy (Appendix 1 a). The respiration rate in animals of Group II was also less than the control till 144 hours of observation. The changes in mean values from 96 to 144 hours were found to be not significant from one another. A similar decreasing trend ($P < 0.05$) was also observed in mean respiration rate of the animals in Group III till 192 hours after fluid therapy. The mean values were found to be variably significant ($P < 0.05$) during the entire period of observation (Appendix 3 a). The mean levels in all the groups were found to be significantly ($P < 0.01$) less than the control level after induction of fluid therapy.

The respiratin was slow and shallow and on ascultation of the chest an involuntary grunting sound was heard in Group I and Group II animals.

Temperature:

The mean rectal temperature of the control animals of all groups was recorded to be between 101.50 ± 0.24 to 102.17 ± 0.14 °F (Table 3). The mean level gradually decreased till 120 hours in Group I. The decrease at 72 hours was not significant than the control. Similarly, the values at 96 and 120 hours were recorded to be not significant between each other, but were significantly ($P < 0.05$) less than the control (Appendix 1 a). The mean level in Group II animals also increased at 72 hours and threafter, dropped till 144 hours. However, the changes up to 120 hours were not significant. The mean value at 144 hours was significantly ($P < 0.05$) lesser than the values of the control at other intervals of time (Appendix 1 a, 2 a, 3 a). The mean values of rectal temperature in animals of Group III were found to be not significant than the control level up to 168 hour. However, the mean value at 192 hours was significantly ($P < 0.05$) less than the values of the control and the values of 72 to 120 hours (Fig. 4).

Ruminal contraction:

The mean ruminal contraction rate was recorded to vary from 7.33 ± 0.27 to 7.67 ± 0.27 per 5 minutes in control animals (Table 3). The rate of contraction significantly ($P < 0.05$) dropped 72 hours after obstruction with the value of 2.33 ± 0.27 per minute in Group I. Thereafter, it increased significantly ($P < 0.05$) till 120 hours of observation following fluid therapy. However, the mean values at 120 hours was significantly ($P < 0.05$) less than the control value. The mean rate of contraction also dropped in all the animals of Group II and the trend was same as in Group I at 72 hours after obstruction. It increased gradually after fluid therapy and significantly ($P < 0.01$) at 120 hours. However, the mean value dropped at 144 hours which was significantly ($P < 0.05$) higher than the values at 72 and 96 hours and significantly ($P < 0.05$) less than the control period.

The mean rate also dropped significantly ($P < 0.05$) at 72 hours of observation in Group III animals. The

Table 3 Showing changes (Mean±SE) in Respiration rate, Rectal temperature and Ruminal contraction before and after pyloric obstruction and after fluid therapy in experimental calves

Parameters	Groups	Before obstruction 24 =	After obstruction HOURS 72	I.V. fluid 96	120	144	168	192
Respiration (per minute)	I	21.33 ± 0.27^{d}	15.33 ± 0.27^{c}	13.00 ± 0.47^{b}	11.00 ± 0.47^{a}	-	-	-
	II	24.00 ± 0.47^{c}	16.67 ± 0.27^{b}	13.33 ± 1.19^{ab}	12.00 ± 1.25^{a}	10.33 ± 0.72^{a}	-	-
	III	23.33 ± 1.19^{d}	16.33 ± 0.27^{c}	14.33 ± 0.72^{bc}	13.00 ± 4.70^{b}	12.00 ± 0.00^{ab}	11.66 ± 0.27^{ab}	10.67 ± 0.27^{a}
Temperature (°F)	I	102.17 ± 0.14^{b}	101.77 ± 0.12^{b}	100.47 ± 0.24^{a}	100.27 ± 0.51^{a}	-	-	-
	II	101.50 ± 0.24^{b}	102.00 ± 0.41^{b}	101.50 ± 0.62^{b}	101.33 ± 0.27^{b}	99.67 ± 0.27^{a}	-	-
	III	101.63 ± 0.59^{b}	101.17 ± 0.36^{b}	100.33 ± 0.20^{ab}	100.93 ± 0.43^{b}	100.83 ± 0.40^{b}	100.20 ± 0.50^{ab}	98.67 ± 0.72^{a}
Ruminal contraction (per minute)	I	7.67 ± 0.27^{c}	2.33 ± 0.27^{a}	3.67 ± 0.54^{b}	4.00 ± 0.00^{b}	-	-	-
	II	7.67 ± 0.27^{c}	2.33 ± 0.27^{a}	3.00 ± 0.42^{a}	5.00 ± 0.47^{b}	4.67 ± 0.27^{b}	-	-
	III	7.33 ± 0.27^{c}	3.67 ± 0.27^{a}	4.33 ± 0.27^{ab}	5.00 ± 0.42^{b}	5.00 ± 0.42^{b}	4.67 ± 0.27^{ab}	4.33 ± 0.27^{ab}
		(3)	(3)	(3)	(3)	(3)	(3)	(3)

= : Control, Figures in the parantheses indicate number of animals

Group treatment: as in the foot note of Table-2

Identical superscripts indicate not significant

Different superscripts indicate significant ($P < 0.05$)

Table 3 Showing changes (Mean±SE) in Respiration rate, Rectal temperature and Ruminal contraction before and after pyloric obstruction and after fluid therapy in experimental calves

Parameters	Groups	Before obstruction 24 =	After obstruction HOURS 72	I.V. fluid 96	120	144	168	192
Respiration (per minute)	I	21.33 ± 0.27^{d}	15.33 ± 0.27^{c}	13.00 ± 0.47^{b}	11.00 ± 0.47^{a}	-	-	-
	II	24.00 ± 0.47^{c}	16.67 ± 0.27^{b}	13.33 ± 1.19^{ab}	12.00 ± 1.25^{a}	10.33 ± 0.72^{a}	-	-
	III	23.33 ± 1.19^{d}	16.33 ± 0.27^{c}	14.33 ± 0.72^{bc}	13.00 ± 4.70^{b}	12.00 ± 0.00^{ab}	11.66 ± 0.27^{ab}	10.67 ± 0.27^{a}
Temperature (^{o}F)	I	102.17 ± 0.14^{b}	101.77 ± 0.12^{b}	100.47 ± 0.24^{a}	100.27 ± 0.51^{a}	-	-	-
	II	101.50 ± 0.24^{b}	102.00 ± 0.41^{b}	101.50 ± 0.62^{b}	101.33 ± 0.27^{b}	99.67 ± 0.27^{a}	-	-
	III	101.63 ± 0.59^{b}	101.17 ± 0.36^{b}	100.33 ± 0.20^{ab}	100.93 ± 0.43^{b}	100.83 ± 0.40^{b}	100.20 ± 0.50^{ab}	98.67 ± 0.72^{a}
Ruminal contraction (per minute)	I	7.67 ± 0.27^{c}	2.33 ± 0.27^{a}	3.67 ± 0.54^{b}	4.00 ± 0.00^{b}	-	-	-
	II	7.67 ± 0.27^{c}	2.33 ± 0.27^{a}	3.00 ± 0.42^{a}	5.00 ± 0.47^{b}	4.67 ± 0.27^{b}	-	-
	III	7.33 ± 0.27^{c}	3.67 ± 0.27^{a}	4.33 ± 0.27^{ab}	5.00 ± 0.42^{b}	5.00 ± 0.42^{b}	4.67 ± 0.27^{ab}	4.33 ± 0.27^{ab}
		(3)	(3)	(3)	(3)	(3)	(3)	(3)

= : Control, Figures in the parantheses indicate number of animals
Group treatment: as in the foot note of Table-2
Identical superscripts indicate not significant
Different superscripts indicate significant ($P < 0.05$)

rate increased after introduction of fluid therapy till 144 hours which subsequently showed a decreasing trend upto 192 hours. The changes in the mean values at all the intervals of this group after fluid therapy were not significant from one another. The mean levels in all the groups were found to be significantly ($P \angle 0.01$) lesser than the control level after introduction of fluid therapy. (Appendix 1 a, 2 a, 3 a and Fig. 5)

The ruminal contraction although increased after fluid, but the force of contraction was found to be weak on palpation in all the group of animals.

Packed Cell Volume:

The mean packed cell volume percentage of the control animals in all the three goups varied between 28.17 ± 0.36 and 29.83 ± 0.59 per cent (Table 4). There was a significant ($P \angle 0.05$) increase from the mean control value reaching the maximum of 48.33 ± 1.60 per cent at 72 hours. The values showed a decreasing trend after administration of Fluid No. 1 reaching a level of 41.33 at 120 hours which was significantly ($P \angle 0.05$) higher than the control and less than the values at 72 hours. However, the mean values at different intervals following fluid therapy did not show any significant change. The animals of group II showed significant ($P < 0.05$) increase in PCV at 72 hours. However, the mean values thereafter significantly ($P < 0.05$) decreased following fluid therapy. The values during the period of fluid therapy were not significantly different from the control values of 35.67 ± 0.54 per cent. The mean values of packed cell volume in Group III animals showed a significant ($P < 0.05$) increase at 72 hours reaching a level of 34.23 ± 0.59 per cent and thereafter showed a significant fall after fluid administrations reaching 28.92 ± 0.45 per cent at 192 hours. The mean values from 96 to 168 hours were recorded to be not significant between the intervals but were significantly ($P < 0.05$) less than the values at 72 hours and more than the control period. However, the mean values at 192 hours were in the range of control period but was significantly ($P < 0.05$) less than the value at 72 to 144 hours. (Fig. 6).

Haemoglobin:

The mean haemoglobin levels in the control animals of all the groups ranged between 9.63 ± 0.33 and 10.83 ± 0.49 gm per cent (Table 4). The mean values at different intervals in Group I showed significant ($P < 0.01$) increase from the control (Appendix 1 b). The highest mean value of 14.20±0.47 gm per cent was recorded at 72 hours post-obstruction. Although, the values decreased gradually after fluid therapy (Fig. 7) the change was not statistically significant during the period of fluid therapy. The mean value at 72 hours in Group II animals was found to be significantly ($P < 0.05$) higher than the control. The values at different intervals after fluid therapy decreased significantly ($P < 0.05$) in comparison to the mean value at 72 hour and was in the range of the control value (Fig. 7). Analysis of variance of the data showed a significant ($P < 0.05$) change in the mean values (Appendix 2 b) of the animals of Group III. The mean value of group III animals at 72 hours was recorded to be significantly ($P < 0.05$) higher than the control as was observed in Group I and Group II. The mean values at different intervals during fluid therapy though showed a decreasing tendency in comparison to the values at 72 hours post-ligation, the values were not significantly different either from those of control or at 72 hours (Fig. 7).

Blood Glucose:

The mean level of blood glucose in the control animals of all the three groups varied between 42.55 ± 0.61 to 46.14 ± 2.01 mg/dl (Table 4). Although, the mean values of animals in group-I showed a decreasing tendency at 72 hours, it was not statistically significant from the control value. However, the blood glucose level increased significantly ($P < 0.05$) during the period of fluid therapy with the highest value of 103.33 ± 1.33 mg/dl at 120 hours. Analysis of variance revealed a significant ($P < 0.01$) increase in the levels of blood glucose (Appendix 1 b) after pyloric obstruction. The animals in Group II also showed a decreasing tendency in blood glucose level as in group-I at 72 hours post-obstruction. There was also a significant increase ($P < 0.05$) at different intervals during fluid therapy reaching the mean level of 149.11 ± 3.66 mg/dl at 144 hour (Appendix 2 b). There was no significant change in blood glucose level till 96 hour in Group III animals but subsequently it significantly ($P < 0.05$) increased reaching a maximum level of 197.30 ± 22.70 mg/dl at 192 hours. The change in values at 120 hours to 192 hours were however not

Table 4 Showing changes (Mean ± S.E.) in packed cell volume, Haemoglobin and Blood glucose before and after pyloric obstruction and after fluid therapy in experimental calves

Parameters	Groups	Before obstruction	After obstruction					
			H	O	U	R	S	
		24 =	72 I.V.fluid	96	120	144	168	192
Packed cell volume (%)	I	29.83±0.59^{a}	48.33±1.60^{c}	43.30±1.96^{bc}	41.33±1.70^{b}	-	-	-
	II	29.33±0.72^{a}	35.67±0.54^{b}	32.00±1.25^{a}	32.00±0.82^{a}	31.87±0.71^{a}	-	-
	III	28.17±0.36^{a}	34.23±0.59^{c}	31.00±0.47^{b}	31.00±0.47^{b}	30.33±1.52^{b}	29.67±1.06^{ab}	28.92±0.45^{a}
Haemoglobin (gm %)	I	10.83±0.49^{a}	14.20±0.47^{b}	12.93±0.43^{b}	12.87±0.38^{b}	-	-	-
	II	10.67±0.39^{a}	12.13±4.04^{b}	10.83±0.36^{a}	10.83±0.14^{a}	10.13±0.15^{a}	-	-
	III	9.63±0.33^{a}	11.43±0.31^{b}	10.33±0.14^{ab}	10.40±0.24^{ab}	10.50±0.82^{ab}	10.00±0.49^{ab}	9.87±0.47^{ab}
Blood glucose mg/dl	I	42.55±0.61^{a}	39.39±0.71^{a}	97.98±1.73^{b}	103.33±1.36^{c}	-	-	-
	II	45.23±1.06^{a}	40.53±0.31^{a}	55.46±1.72^{b}	115.90±2.40^{c}	149.11±3.66^{d}	-	-
	III	46.14±2.01^{a}	44.37±6.92^{a}	68.41±13.40^{a}	150.51±26.29^{b}	171.28±25.27^{b}	188.16±22.10^{b}	197.30±22.74^{b}
		(3)	(3)	(3)	(3)	(3)	(3)	(3)

= : Control,

Figures in parenthesis indicate number of animals
Group treatment: As in the foot note of Table 2
Identical superscripts indicate not significant
Different superscripts indicate significant

not significant. The mean levels in all the groups were found to be significantly (P∠ 0.01) higher than the control level after introduction of fluid therapy (Appendix 1 b, 2 b, 3 b and Fig 8).

<u>Calcium:</u>

The mean calcium values of the control animals were found to be ranging between 9.57 ± 0.19 and 10.08 + 0.28 mg/dl (Table 5). Analysis of variance revealed that the values at different intervals during the period of investigation in Group I were not significant from one another. Similar observation was also made in animals of Group II and Group III (Fig.9) according to Appendix 1 b, 2 b, 3 b.

<u>Potassium:</u>

The mean potassium level of the control animals of all the groups was recorded to be ranging between 5.01 ± 0.01 and 5.08 ± 0.08 mEq./L. (Table 5). Significant increase (P∠ 0.05) was observed at 72 hours in animals of group-I. It showed an increasing trend after infusion of fluid reaching the level of 4.12 ± 0.18 mEq./L. at 120 hours. However, the increasing trend from 72 to 120 hour were not significant as revealed from the analysis of variance (Appendix 1 b). Similar trend has been recorded in the animals of Group II also. Analysis of variance showed a significant ($P \angle 0.05$) change in the mean values (Appendix 2 b). There was significant ($P \angle 0.05$) drop in the mean potassium level in animals of Group II at all the intervals of investigation than the control value (Table 5). The value showed significant ($P \angle 0.05$) change from one another during all the intervals of fluid therapy (Fig. 10) as per the Appendix 3b.

Sodium:

The mean level of Sodium during control period of all the three groups varied between 135.29±5.31 and 144.33 ± 0.98 mEq./L. (Table 5). Analysis of variance revealed that sodium levels of all the three groups of animals at different intervals of investigation did not show any sifnificant change (Appendix 1c, 2c, 3c) between one another. (Fig. 11).

Chloride:

The mean chloride levels during the control period in all the three groups varied between 105.78 ± 3.12 and 113.00 ± 0.85 mEq./L. (Table 6). Analysis variance revealed significant ($P \angle 0.01$) fall at different intervals of investigation from the control levels in Group I and

Table 5 Showing changes (Mean ± S.E.) in Serum Calcium, Potassium and Sodium before and after pyloric obstruction and after fluid therapy in experimental calves

Parameters	Groups	Before obstruction	After obstruction					
		24 =	72 I.V. fluid	96	120	144	168	192
Calcium mg/dl	I	9.57 ± 0.19^{a}	9.68 ± 0.30^{a}	10.12 ± 0.13^{a}	9.93 ± 0.25^{a}	-	-	-
	II	10.08 ± 0.28^{a}	10.56 ± 0.19^{a}	10.46 ± 0.24^{a}	10.46 ± 0.29^{a}	10.42 ± 0.21^{a}	-	-
	III	9.70 ± 0.21^{a}	10.40 ± 0.28^{a}	9.77 ± 0.14^{a}	10.27 ± 0.33^{a}	9.68 ± 0.28^{a}	9.99 ± 0.53^{a}	9.00 ± 0.45^{a}
Potassium mEq/L.	I	5.01 ± 0.01^{b}	4.00 ± 0.02^{a}	4.06 ± 0.10^{a}	4.12 ± 0.18^{a}	-	-	-
	II	5.03 ± 0.44^{b}	4.08 ± 0.27^{a}	4.26 ± 0.11^{a}	4.36 ± 0.23^{a}	4.26 ± 0.19^{a}	-	-
	III	5.08 ± 0.08^{c}	3.51 ± 0.20^{a}	3.82 ± 0.28^{ab}	4.30 ± 0.19^{b}	4.09 ± 0.14^{ab}	4.04 ± 0.18^{ab}	3.98 ± 0.06^{ab}
Sodium mEq/L.	I	144.33 ± 0.98^{a}	141.00 ± 0.94^{a}	141.33 ± 1.09^{a}	141.00 ± 0.24^{a}	-	-	-
	II	144.03 ± 1.08^{a}	142.80 ± 1.10^{a}	142.80 ± 0.34^{a}	141.78 ± 1.02^{a}	143.23 ± 1.02^{a}	-	-
	III	135.29 ± 5.31^{a}	132.83 ± 6.01^{a}	133.24 ± 5.24^{a}	131.98 ± 4.88^{a}	133.88 ± 5.27^{a}	132.82 ± 4.62^{a}	131.98 ± 4.51^{a}
		(3)	(3)	(3)	(3)	(3)	(3)	(3)

= Control, Figures in parenthesis indicate number of animals
Group treatment: as in the foot note of Table 2.
Identical superscripts indicate not significant.
Different superscripts indicate significant ($P < 0.05$)

Group II whereas the change was significant ($P < 0.05$) in Group III, (Appendix 1 c, 2 c, 3 c). However, the change in values at different intervals during fluid therapy were not statistically significant from the mean values at 72 hours (Fig. 12). Similar observations have been made in all the three groups of animals.

Blood urea nitrogen:

The mean blood urea nitrogen in all the three groups during control period ranged between 6.35 ± 0.08 and 11.67 ± 0.72 mg/dl.(Table 6). Analysis of variance revealed a significant increase ($P < 0.01$) in blood urea nitrogen level from the control values (Appendix 1 c, 2 c, 3 c). In group I, the mean BUN values increased significantly ($P < 0.05$) after pyloric obstruction and the increase continued even after fluid therapy reaching a highest value of 132.86 ± 5.26mg/dl at 120 hours. However, the change in levels at 72 and 96 hours were not significant. Similar trend as in Group I was also observed in Group II (Fig. 13) except that the values at 96 hours and 120 hours were not differing significantly and reaching a highest level of 146.69 ± 2.86 mg/dl at 144 hours.

In Group III animals the blood urea nitrogen level showed an increasing trend after pyloric obstruction.

It continued as such even after institution of fluid therapy and touched a maximum level of 45.67 ± 4.00 mg/dl. at 192 hours post-obstruction and fluid therapy. The increasing trend was significant ($P < 0.05$) upto 144 hours and thereafter, no significant change was recorded.

Creatinine:

The mean creatinine values of the control period in all the three groups varied between 2.08 ± 0.05 and 2.25 ± 0.11 mg/dl. (Table 6). There was a significantly ($P < 0.01$) decreasing trend in the level of creatinine after obstruction and after fluid therapy in all the groups (Appendix 1 c, 2 c, 3 c). The levels at different intervals of fluid therapy in Group I were found to be significantly ($P < 0.05$) less than the value of control period, but was not significant during fluid therapy. Similarly, a significantly ($P < 0.05$) decreasing trend was also observed in Group II animals at different hours of post-obstruction and fluid therapy than the control value. The values after fluid therapy were having a variably significant ($P < 0.05$) decrease than one another.

In Group III animals there was significant ($P < 0.05$) drop of the creatinine level at differsnt

Table 6 Showing changes (Mean ± S.E.) in Serum Chloride, Urea nitrogen and Creatinine before and after pyloric obstruction and after fluid therapy in experimental calves

Parameters	Groups	Before obstruction	After obstruction					
		HOURS						
		24 =	72 I.V.fluid	96	120	144	168	192
Chloride mEq/L.	I	113.00±0.85^{b}	90.80±0.91^{a}	90.75±0.51^{a}	91.85±1.58^{a}	-	-	-
	II	109.50±0.85^{b}	80.60±0.93^{a}	82.33±1.34^{a}	83.03±1.24^{a}	83.00±1.63^{a}	-	-
	III	105.78±3.12^{b}	79.65±4.16^{a}	80.36±4.02^{a}	80.57±3.67^{a}	81.67±6.15^{a}	80.73±5.46^{a}	79.92±5.77^{a}
Urea nitrogen mg/dl.	I	11.67±0.72^{a}	97.93±0.36^{b}	104.73±2.19^{b}	132.86±5.26^{c}	-	-	-
	II	10.17±1.06^{a}	53.43±1.28^{b}	90.28±0.26^{c}	113.03±1.19^{c}	146.69±2.86^{d}	-	-
	III	6.35±0.08^{a}	9.88±0.29^{a}	17.35±5.19^{b}	22.79±1.37^{c}	41.71±0.73^{d}	44.81±3.08^{d}	45.67±4.00^{d}
Creatinine mg/dl	I	2.08±0.05^{c}	1.72±0.04^{b}	1.54±0.02^{ab}	1.35±0.04^{a}	-	-	-
	II	2.25±0.11^{d}	1.93±0.58^{c}	1.74±0.05^{c}	1.51±0.05^{ab}	1.28±0.08^{a}	-	-
	III	2.20±0.05^{e}	1.94±0.03^{a}	1.87±0.05^{d}	1.74±0.03^{c}	1.62±0.03^{c}	1.42±0.05^{b}	1.05±0.02^{a}
		(3)	(3)	(3)	(3)	(3)	(3)	(3)

= Control Figures in parenthesis indicate number of animals

Group treatment: : as in foot note of Table 2.

Identical superscripts indicate not significant

Different superscripts indicate significant (P ∠ 0.05) hours of post-obstruction and fluid therapy reaching a maximum value of 1.05 $\pm$ 0.02mg/dl . at 192 hours. The levels at 72 and 96 hours were not significant from each other. Similarly, the level at 120 and 144 hours did not show any significant change (Fig. 14).

* * * * *

CHAPTER V

DISCUSSION

D I S C U S S I O N

Prior precise diagnosis and resuscitation of the animal with correct fluid to bring the body homoeostasis to its near normal condition are the two major preoperative procedures. These are to be followed before to surgical manupulation of cases in pyloric obstruction. Sufficient time is not allowed for accurate diagnosis and treatment due to the acuteness of this malady and the affected animal may not survive more than 5 days Pathy (1986). In order to minimise the surgical risk preoperative treatment with appropriate fluid is highly essential in preparing the animal for definite surgeory.

Pyloric obstruction was created through right lower paracostal approach as has been suggested by Papadopoulos et al. (1985 a), Pathy (1986), Basu (1987), Mishra (1987), Braun et al. (1990) and Sethy (1990).

Sedation with Triflupomazine and local

infiltration with 2% Lignocaine with adrenaline (Lignox 2% marketed by Warren Pharmaceuticals Ltd. Bombay) in two tiers proved satisfactory for surgical manupulation. The animal was restrained throughout the period of operation on left lateral recumbency.

The right lower paracostal area was selected because the abomasum lies extended below the 7th to 8th rib (Anderson and Anderson, 1980). The pylorus is located opssite to 9th and 10th rib. This approach provided sufficient space for easy munupulation of the abomasum and pylorus. The pylorus was exteriorised slightly posteriorly. (Plate 1). Blood vessels were avoided while ligating the pylorus to simulate the clinical cases.

Umbilical tape was used in the present experiment to ligate the pylorus, Pathy (1986), Basu(1987) and Sethy (1990) used the same material for creation of pyloric obstruction. Though other ligating materials like flexible plastic tubing Papadopoulos et al. (1985 a) and venyl plastic tubing with cotton tape Hammond et al.(1964) and Corker and Dziuk (1968) were used earlier, use of umbilical tape for ligation and obstruction of pylorus gave satisfactory result and did not damage the wall of the pylorus.

Symptoms:

All the animals developed gradual reduction in appetite with dysphagia and complete anorexia at 72 hours after pyloric obstruction. In the opinion of Blood et al. (1985) that reduction in appetite with dysphagia is a common sign in any alimentary tract stasis due to any cause. The animals of all the groups accepted food up to 96 hours following fluid therapy. This may be due to correction of electrolyte imbalance and dehydration and provision of nourishment in the therapeutic fluids. Complete anorexia after 96 hours in all the animals may be due to over distension of rumen and abomasum with ingesta and gastric secretions. Reduction of appetite was also observed by Pinsent (1962), Corker and Dziuk (1968), Papadopoulos et al. (1985 a), Kuiper and Breukink (1986 a), Pathy (1986) and Basu (1987) in cases of pyloric obstruction.

Continued dehydration even during fluid therapy was observed and was evinced by increased PCV (Table 4). Similar observation was made by Pathy(1986).

Rumination was suspended in the animals of all the three groups within 24 hours of ligation of pylorus till death. This might be due to the pain caused by obstruction to the pylorus (Papadopoulos et al. (1985 a) or damage caused by ligation to the branches of the vagus supplying to the pylorus Hoflund (1940) and Neal and Edwards (1968).

When viewed from behind a 'L' shaped abdominal distension was noticed in all the animals at 72 hours after pyloric obstruction. This was due to distension of rumen on the left and abomasum on the right side caused by accumulation of undigested food materials alongwith water and gastric secretions, ligation of pylorus did arrest the passage of food materials into the intestinal tract. The characteristic 'L' shaped distension of the rumen along with the distension of right lower abdomen has also been reported by Whitlock (1976), Ferrante and Whitlock (1981) also tormed it as Papple shaped distension. Such characteristic abdominal distension has also been observed by Naerland and Helle (1962), Pinsent (1962) Esperson (1964) Neal and Edwards (1968), Kuiper and Breukink (1986 a), Pathy (1986), Basu (1987) and Sethy (1990). The accumulation of fluid in the rumen could be ascribed to contineous in take of water inspite of inappetance (Hoflund, 1940) and accumulation of saliva and mainly reflux of abomasal fluid in to the rumen. (Papadopoulos et al. 1985 a and Basu, 1987).

All the animals in this experiment exhibited the characteristic sign of dehydration such as sunken eyes and dry vesible mucous membrane and dry muzzle at 72 hours post ligation. The dehydration is due to accumulation of fluid in the rumen, which could not be obsorbed into the circulation due to pyloric obstruction. Moreover fluid loss is continued in the form of evaporation, urination and secretions of saliva and gastric juice. Similar views have been expressed by Rathor et al. (1977), Dass et al.(1981 a) Ashcroft (1983) Papadopoulos et al. (1985 a), Basu (1987) and Mishra (1987).

The splashing sounds heard on ballottment in the present experimental animals after 72 hours suggested the accumulation of large quantity of fluid in the rumen and abomasum. This was also observed by Whitlock (1969) Dass et al. (1981 a), Ferrante and Whitlock (1981), Pathy (1986), Basu (1987), Mishra (1987) and Sethy (1990). The deep resonance on percussion on the left flank can be attributed to trapping of gas in the upperportion of the dorsal sac of rumen.

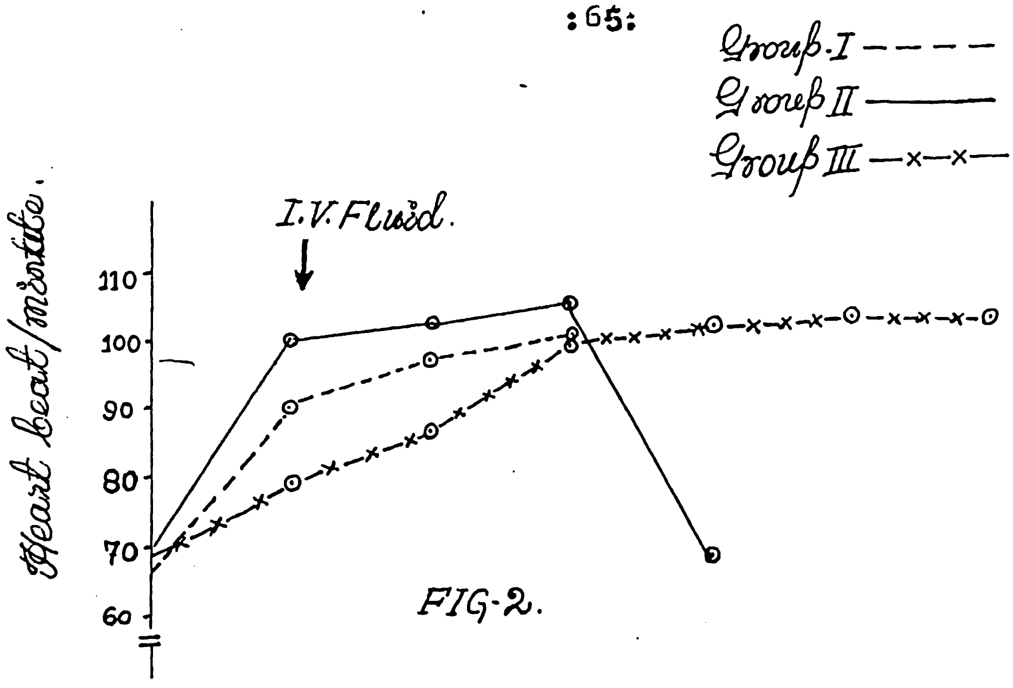
All the experimental animals voided scanty, dry and pelleted faeces which stopped after 72 hours of obstruction. The ligation of pylorus prevented the onward passage of ingesta across the pylorus. As such the limited amount of food material passed to the intestine prior to the ligation has been voided till 72 hours after ligation. The dry and hard consistency of the faeces in the later stages was due to the prolonged transit time in the intestine (Whitelock (1976) and dehydration.

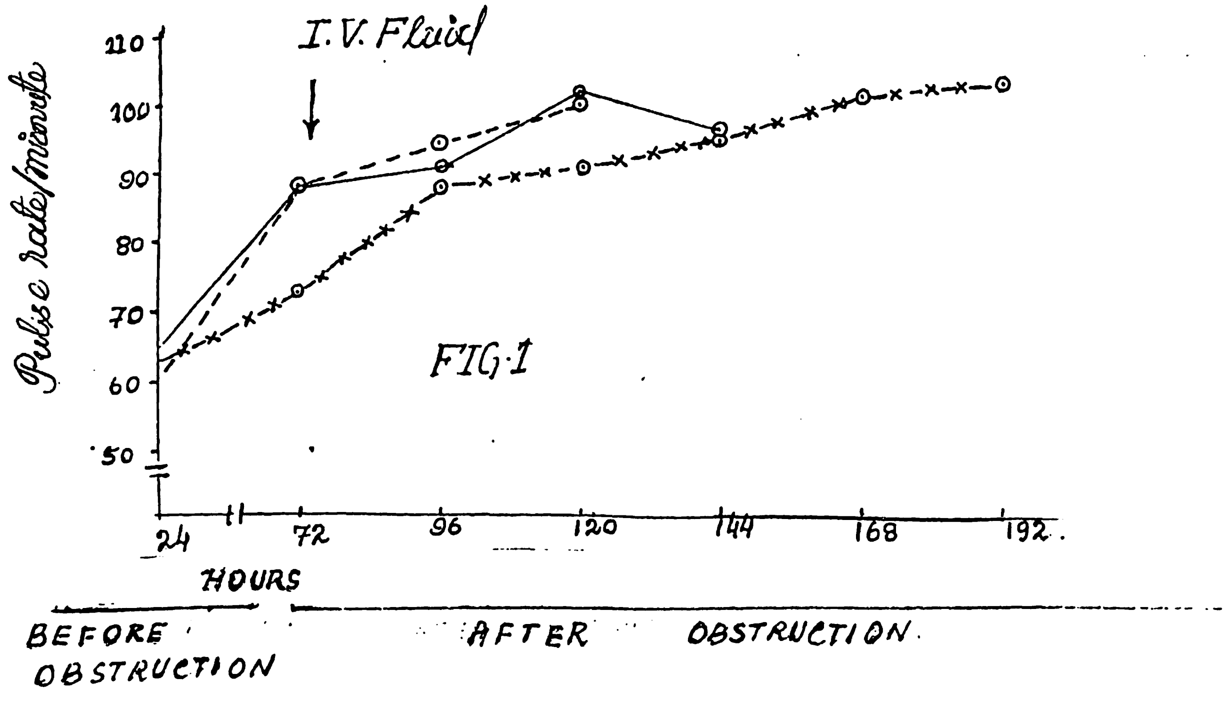
Apparent reduction in the volume of urine of the experimental animals was due to dehydration as evident through increase haemoconcentration. Similar views were expressed by Papadopoulos et al. (1985 a) and Basu (1987).

The pulse rate in the animals of all the groups prior to ligation of pylorus was ranging between 60.67 ± 0.54 and 65.00 ± 0.90 per minute in the present experiment which is in the normal range. Blood et al. (1985) have stated that the normal range of pulse rate is 68-80 per minute. Significant ($P < 0.05$) rise in pulse rate in all the experimental animals was marked at 72 hours after pyloric obstruction leading to hypovolaemia (Papadopoulos et al., 1985 a, and Basu et al. 1990).

The significant rise ($P < 0.05$) in pulse rate continued even during fluid therapy in Group I and Group II animals. However, the animals in Group III also exhibited a slow and gradual rise in pulse rate from 96 hours onwards (Table 2, Fig.1). This indicated slow deterioration of the condition of the animals in Group III in comparison to Group I and Group II which might be due to less severe dehydration as indicated though less severe change in PCV during fluid therapy of Group III animals.

The rate of heart beat of all the experimental animals before pyloric obstrcution was in the range of 60.67 ± 0.54 and 70.33 ± 0.27 per minute which was in the normal range of 48-84 per minute (Detweiler, 1984). There was a significant ($P < 0.01$) increase in the rate of heart beat in all the three groups of animals at 72 hours after pyloric obstruction (Appendix 1 a, 2 a, 3 a). The increase in rate of heart beat might be due to dehydration and reduction in plasma volume as observed though increase in PCV (Table 4). Similar opinion has also been expressed by Hutchins *et al.* (1957) and Papadopoulos *et al.* (1985 a) in experimental duodenal obstruction in cattle and Basu (1987), Mishra (1987) and Sethy (1990) in experimentally induced pyloric obstruction.





GRAPH SHOWING CHANGES IN PULSE, HEART BEAT BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY

After induction of fluid therapy at 72 hours significant ($P \angle 0.05$) increase in heart rate continued up to 96 hours in Group I and Group III which remained unchanged till 192 hours. However, no significant increase in heart beat was observed in animals of group II following the administration of fluid at 72 hours. But the rate of change in heart beat in animals of group III was marked when compared with other group (Fig.2). This might be due to effect on body metabolism by the fluid III containing the required electrolytes like Sodium, potassium, Calcium, chloride with 20% dextrose. The rise in rate of heart beat in all groups might be due to continued hypovolaemia as also expressed by Basu (1987) and Mishra (1987). The marked change in group III animals during fluid therapy might be due to elevated level of BUN in that group (Table 6). Hutchins et al. (1957), Papadopoulos et al. (1985 a) have recorded a rise in heart rate in experimental duodenal obstruction with increased BUN.

The rate of respiration per minute was found to be ranging between 21.33 ± 0.27 and 24.00 ± 0.47 in all three groups of animal prior to pyloric obstruction which is in the normal range of 10.30 (Blood et al. 1985). There is a significant ($P \angle 0.01$) fall of respiration by 72 hours after ligation in all the groups ranging between 15.33 ± 2.27 and 16.67 ± 0.27 per minute (Appendix 1a, 2a, 3a). The fall in respiration rate at 72 hours might be due to increase in blood pH as suggested by Hypochloraemia (Table 6) which can be collaborated with the opinion of Papadopoulos _et al_. (1985 a and 1985 b). They have stated that the respiratory depression may act as compensatory mechanism to combat the metabolic ackalosis. The respiratory rate decreased further significantly ($P < 0.05$) in Group I and Group II even during fluid therapy and gradual and significant decrease in Group III (Fig. 3) can be attributed to over distension of rumen and abomasum and continued hypochloraemic condition even during fluid therapy.

The body temperature in animals of all the three groups in ranging between 101.50 ± 0.24 and $102.17 \pm 0.14^{o}F$ which is in the normal range of 101.5 to $103^{o}F$ (Blood et al., 1985). The body temperature did not show any significant change in all the groups (Table 3) till the terminal stages. There was a significant ($P < 0.05$) fall of temperature at the terminal stage. The fall in temperature at the terminal stages might be due to dehydration Hypokalaemia and decreased metabolic activity. Pearson (1974), Ascraft (1983), Papadopoulos et al. (1985 a) recorded lower body temperature following intestinal obstruction. In the present experiment the body temperature remains within the normal range which might be due to the contineous supply of glucose through the infusion fluid (Fig. 4).

All the animals had normal ruminal motility before pyloric obstruction which decreased significantly ($P \angle 0.01$) following pyloric obstruction (Appendix 1a, 2a, 3a). The rate of contractions which decreased significantly ($P \angle 0.01$) 72 hours increased gradually and significantly ($P \angle 0.05$) following fluid therapy (Table 3). However, the force of contraction appears to be weak on palpation. The significant ($P \angle 0.01$), fall in the rate of contraction by 72 hours might be due to distension of fore-stomachs, dehydration, electrolyte and acid base imbalance (Table 5.). This corroborates with views of Blood et al. (1985) who have mentioned that a change in body water, acid base and electrolyte balance, alkalosis may lead to reduced or complete cessation of ruminal contraction. Significant ($P \angle 0.05$) rise in rumen contraction following fluid therapy further provded that correction of dehydration and electrolyte balance will improve the ruminal motility. Further, the increase in the rate of rumen motility might be also due to

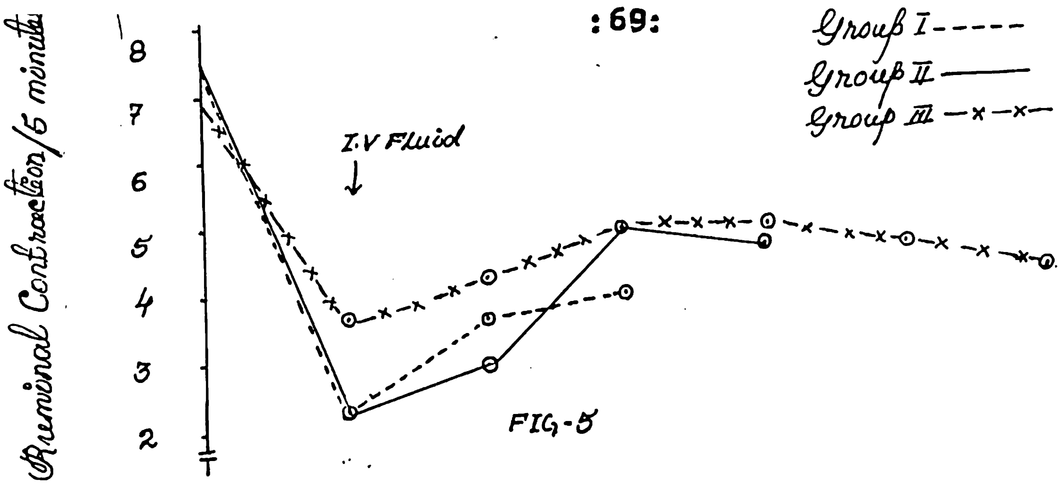


FIG-5

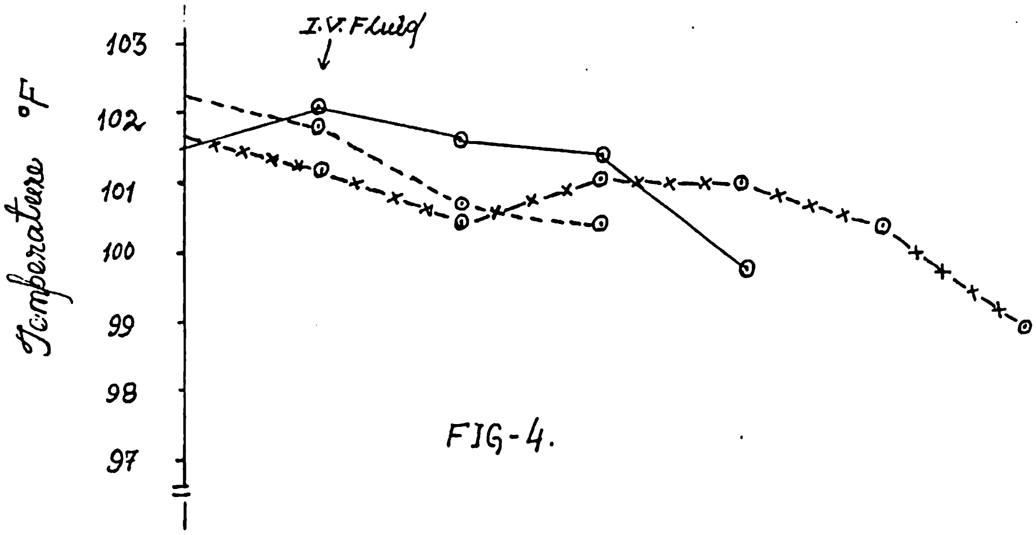


FIG-4.

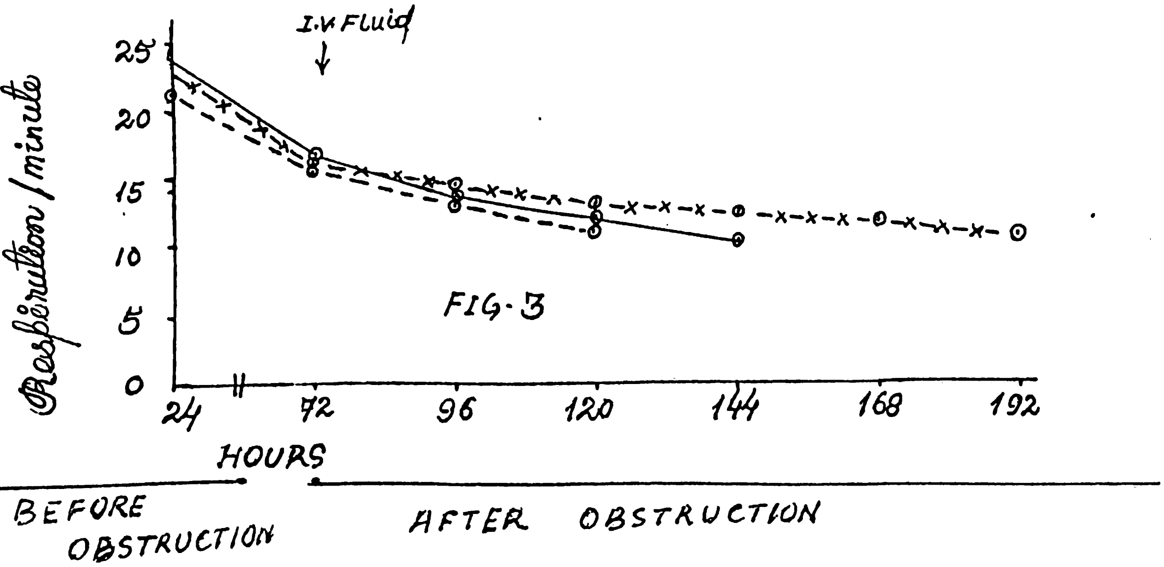


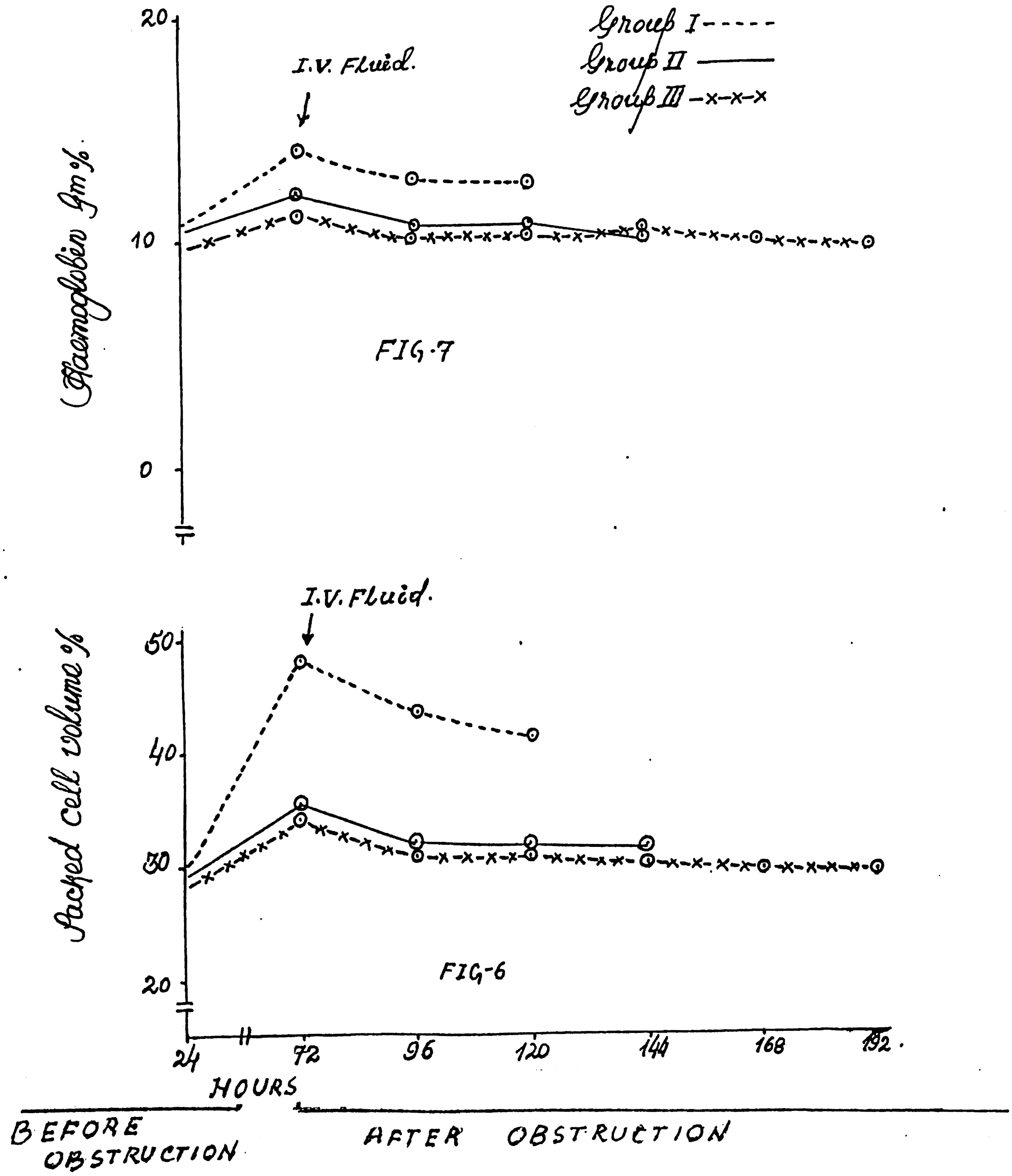
FIG-3

GRAPH SHOWING CHANGES IN RESPIRATION, TEMPERATURE AND RUMINAL CONTRACTION BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY mild stimulation to low tension receptarts in the rumen by the increase in rumen pressure as suggested by Ferrante and Whitlock (1981) an in (Fig. 5).

Analysis of variance of date indicated significant ($P \angle 0.01$) increase in the PCV in Group I and ($P \angle 0.01$) in Group II and Group III animals. There was a significant ($P \angle 0.01$) increase in the haematocrit values by 72 hours following pyloric obstruction in all the three groups. This may be due to dehydration caused by sequestration of fluid in the rumen and abomasum as evidenced by splashing sound on ballottment and continued loss of fluid through evaporation and urine. This is in confirmity with the findings of Poulsen (1974), Smith (1978). Papadopoulos et al. 1985b) who recorded in increase in PCV in obstructive diseases of the intestine. The haematocrit values decreased significantly ($P \angle 0.05$) with 24 hours and remained as such till the terminal stage in Group I and returned to preobstructive values in Group II and Group III (Table 4 and Fig. 6). In Group I PCV did not reach the preoperative value as they could not servive and the longivity is less than the other groups. However, administration of fluid reduced the amount of dehydration in all the three goups and is in confirmity with the findings of Pathy (1986) who did observe in the improvement following fluid therapy.

Change in levels of haemoglobin after pyloric obstruction showed a direct relationship with PCV value. Analysis of variance in all the three groups indicated a significant ($P \angle 0.01$) increase in haemoglobin level of group I and ($P \angle 0.05$) in Group II animals. A significant rise in haemoglobin by 72 hours after obstruction, can be attributed to the same reason responsible for the increased PCV values. The haemoglobin concentration decreased gradually following fluid therapy as it caused haemodilution and is in agreement with the finding of Pathy (1986). However, the concentration of Haemoglobin in Group III animals following fluid therapy are not statistically different from the values of pre and past pyloric obstruction (Table 4, Fig. 7). This might be due to the effect on fluid and electrolyte status provided by Fluid III.

The mean blood glucose level in all the three groups was ranging between 42.55 ± 0.61 and 46.14 ± 2.01 mg/dl and is within the normal range of 35-55 mg/dl documented by Blood et al. (1985). Analaysis of variance indicated a significant ($P \angle 0.01$) change in all the three groups.



GRAPH SHOWING CHANGES IN PACKED CELL VOLUME AND HAEMOGLOBIN IN BLOOD BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY

The mean level of glucose showed a non significant decrease by 72 hours post ligation in all the three groups. However, the level of glucose increased significantly ($P < 0.01$) during the period under fluid therapy, till the terminal stages both in group I and group II whereas, the animals in Group III showed a significant ($P < 0.05$) increase by 120 hours. It did not show any further raise up to 192 hours. The initial fall of blood glucose level in all the three groups might be due to decrease in feed intake and metabolic activity. Subsequent hyperglycaemic condtions can be attributed to dehydration, increased haemoconcentration and severe stress produced by the pressure of distended fore-stomach on different organs (Mills and Chirstian, 1970). The stress might be due to the release of epinephrine. Beckett et al. (1967) leading to hyperglycaemic condition. Pope (1961), Whitlock (1980) have also observed a considerable increase in blood glucose in abomasal impaction. In the present experiment continued administration of fluid containing different level of glucose might have been a contributory factor. In animals of group III the level of blood glucose increased a highest level of 197.30 ± 22.74 mg/dl in comparison to other groups (Fig. 8). It can be attributed to the high level of dextrose 20 percent in the infusion fluid.

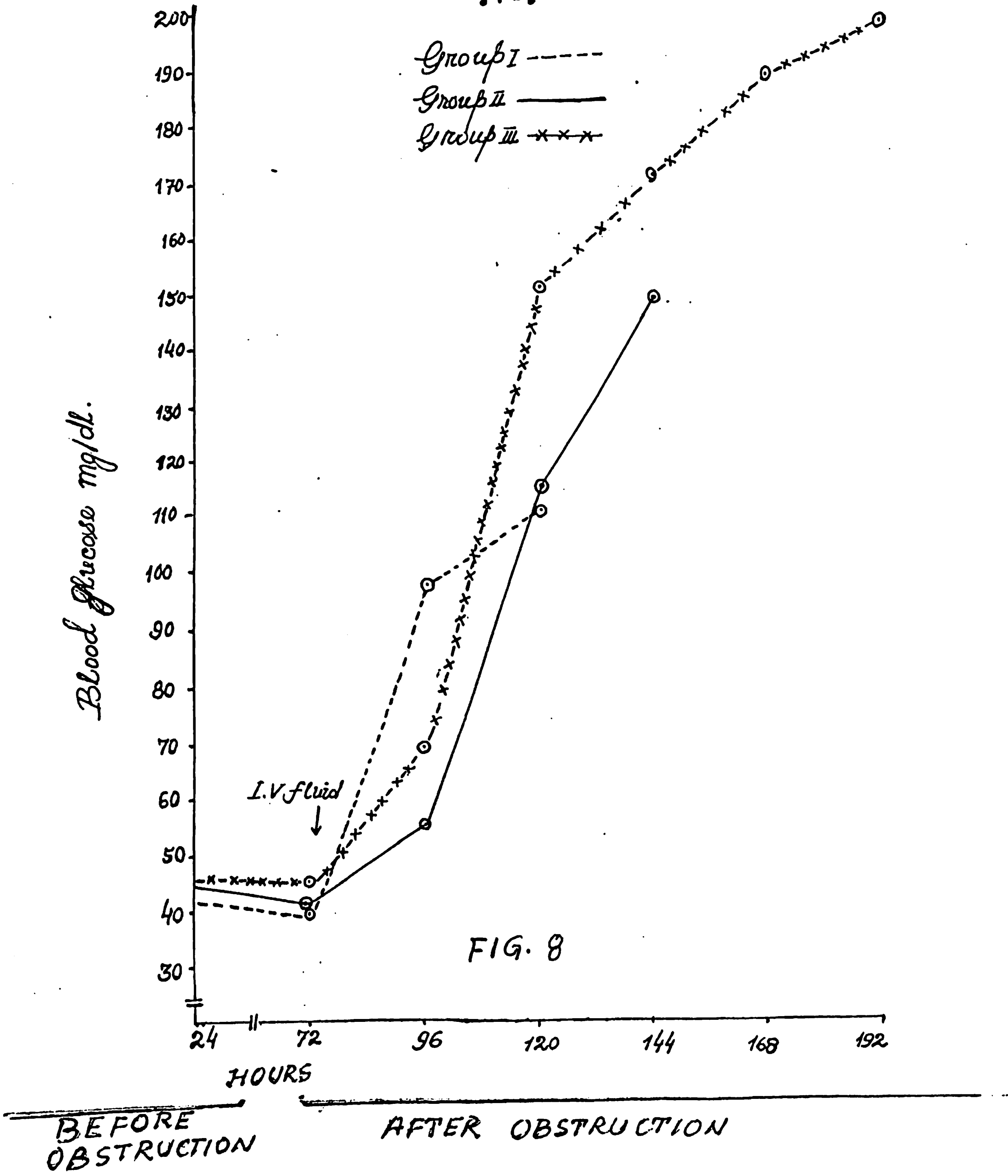
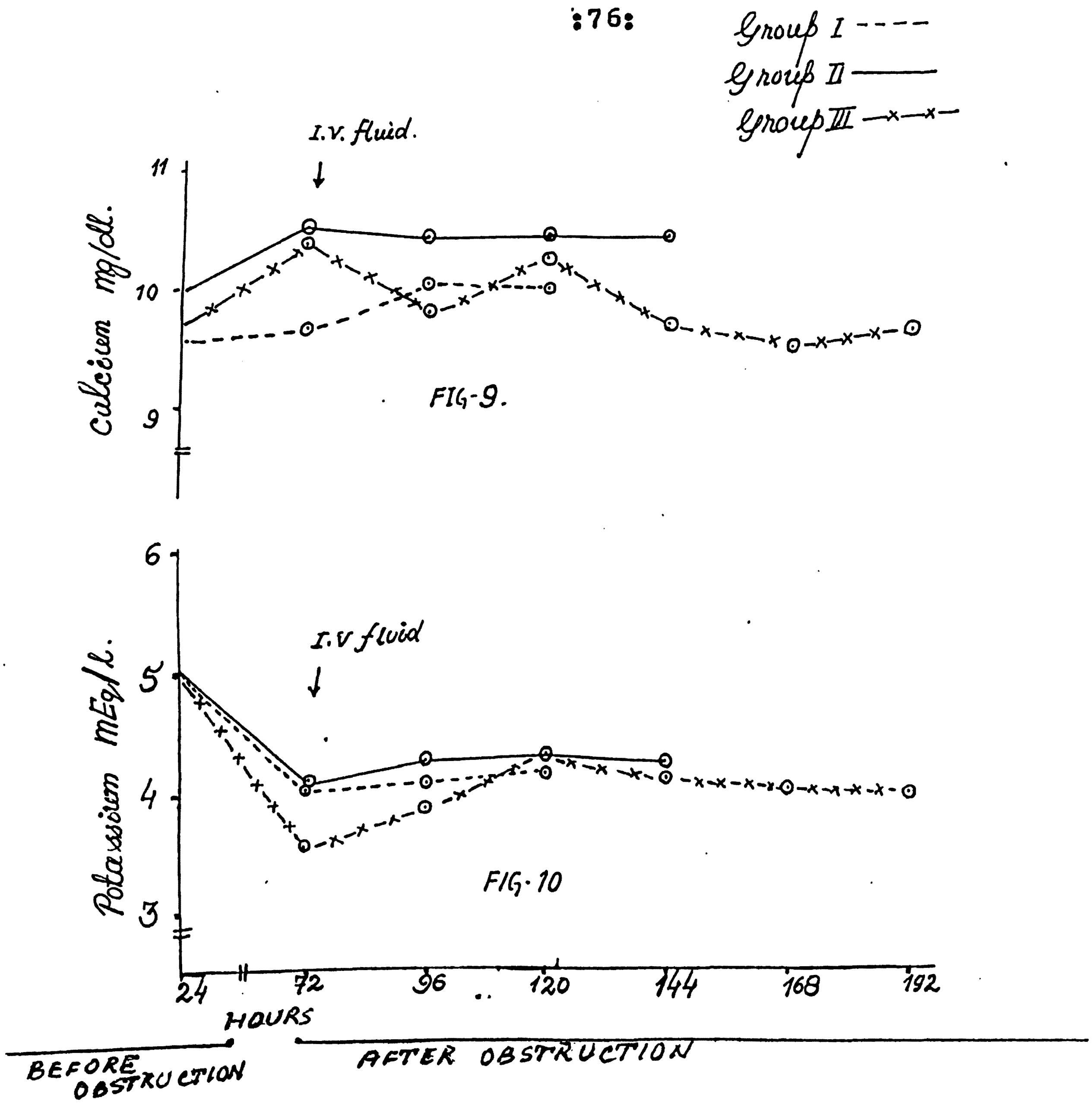


FIG. 8

GRAPH SHOWING CHANGES IN BLOOD GLUCOSE BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY

The mean serum calcium level in all the three groups prior to creation of pyloric obstruction ranged between 9.57 ± 0.19 and 10.08 ± 0.28 mg/dl (Table 5) and is within the normal range of 8.0 to 10.5 mg/dl according the Blood et al.(1985). The level of calcium in all the three groups did not have any significant change during the entire period of investigation (Appendix 1b, 2b, 3b and Fig. 9). Pathy (1986) did observe a decline in calcium level even during the period of fluid therapy in the calves subjected to pyloric obstruction. This can be attributed to low level of calcium included in the transfusion fluid, whereas in the present investigation the level of calcium chloride added was 0.1 % against 0.048% used by Pathy (1986).

The securm potassium level during the control period ranged between 5.01 ± 0.01 and 5.08 ± 0.08 mEq/L. (Table 5) and is within the normal range of 3.9 to 5.8 mEq/L. as stated by Blood et al. (1985). It decreased significantly ($P < 0.01$) following pyloric obstruction in Group I (Appendix 1b) and ($P < 0.05$) in Group II and Group III (Appendix 2b, 3b). The significant fall ($P < 0.01$) following 72 hours pyloric obstruction may be due to alkalosis as evident by decreased level of chlorides (Table 6). Similar changes have also been observed by Hammond et al. (1964),



GRAPH SHOWING CHANGES IN SERUM CALCIUM AND POTASSIUM BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY

Papadopoulos *et al.* (1985 b) and Avery (1986) in experimental duodenal obstruction and Robertson (1965), Dass *et al.* (1981 a) and Lattman (1984), in cases of abomasal idsplacement. The mean serum potassium level did not show any further significant decrease during fluid therapy in comparison to the values at 72 hours post-ligation. Basu (1987) has recorded a continued significant ($P < 0.01$) decrease in potassium level following pyloric obstruction till the terminal stages. The present investigation indicated that addition of Potassium Chloride to the transfusion fluid prevented the further decrease in the potassium level in spite of continued pyloric obstruction in all the three groups (Fig. 10).

The mean serum sodium level in all the three groups ranged between 135.29 ± 5.31 to 144.33 ± 0.98 mEq/L. during the control period and was within the normal range of 132 to 152 mEq/L. (Blood *et al.*, 1985). The serum sodium level did not show any significant change following pyloric obstruction in any of the group. Similarly no significant change could be detected even during the period of fluid therapy. Similar observation was also made by Pathy (1986) and Basu (1987). Even addition of sodium chloride in the transfusion solution did not cause any elevation in the level of sodium. This might be due to continued accumulation of sodium in the fore-stomach through gastric secretions.(Mishra, 1987) and Saliva (Michel, 1985). Moreover, alteration in plasma sodium level in a significant way may not be possible because of availability of bone reservoir both for storage and supply (Morre, 1954).

The mean serum chloride level in all the three groups ranged between 105.78 ± 3.12 and 113.00 ± 0.85 mEq/L. during the control period and was within and bormal range. It decreased significantly (P∠ 0.01) as per Appendix (1c, 2c and 3c) in all the three groups. The significant (P∠ 0.01) decrease was observel by 72 hours past obstruction and there was no further significant decrease during the fluid therapy. The significant (P∠ 0.01) decrease at 72 hours might be due to accumulation of gastric secretion in the abomasum and fore-stomach and failure of its passage through pylorus for its absorption. Similar observation was made by Hammond et al. (1964), Corker and Dziuk (1968), Papadopoulos et al. (1985 b) and Basu (1987) in cases of impaired abomasal outflow. The absence of significant change in serum chloride level following fluid therapy in all the three groups of animals (Fig. 12) indicated that an

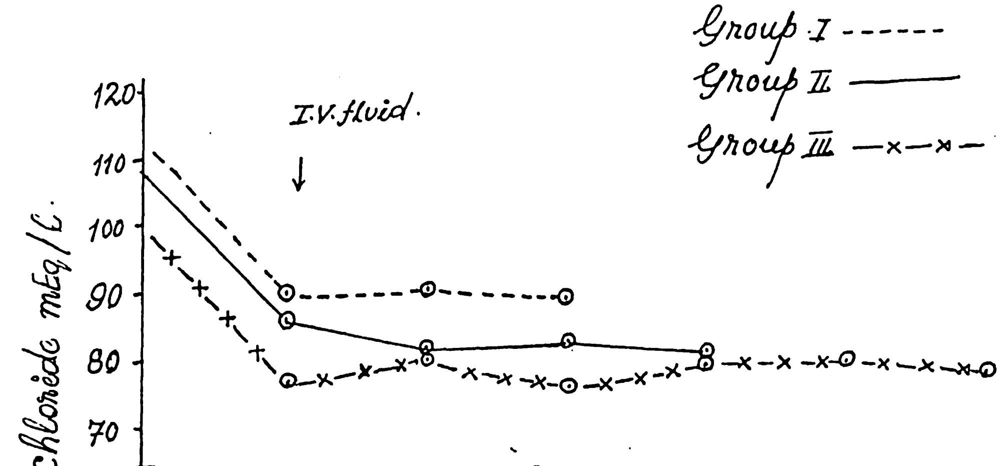


FIG-12

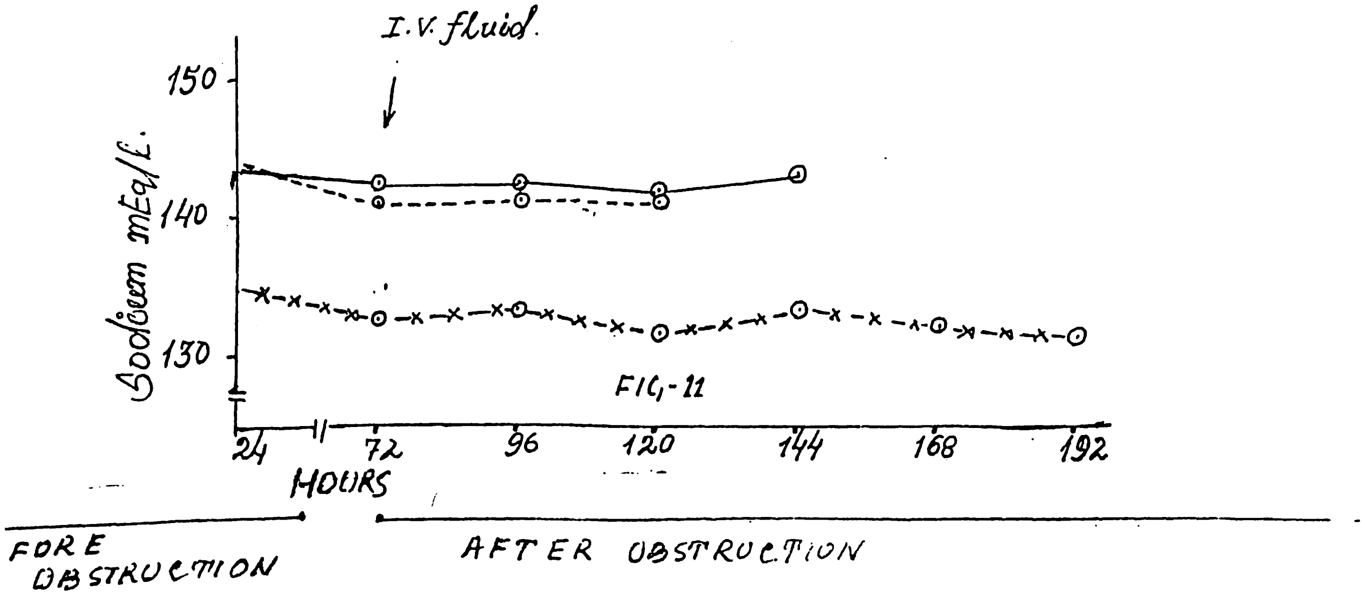


FIG-11

GRAPH SHOWING CHANGES IN SERUM SODIUM AND CHLORIDE BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY.

addition of chloride ions through sodium chloride, potassium chloride and calcium chloride in the transfusion fluid did not raise the chloride level which may be due to continued sequestration of their ion in the abomasum and fore-stomach Mishra (1987). The present data indicted that it may be required to add more chloride ions in the transfusion fluid to bring to the preoperative value.

The mean urea nitrogen level of serum was estimated to be in the range of 6.35 ± 0.08 to 11.67 ± 0.72 mg/dl during the control period. This is in normal range of 6 to 27 mg/dl (Blood et al., 1985). The serum urea nitrogen level showed a significant ($P < 0.01$) change in all the groups of animals (Appendix 1c, 2c, 3c). There was a significant ($P < 0.01$) increase following pyloric obstruction in Group I and Group II at 72 hours and continued to increase significantly ($P < 0.01$) till terminal stages in those groups. In Group III though there was rise in urea nitrogen level at 72 hours, it was not statistically significant. However the level increased still further significantly ($P < 0.05$) up to 144 hours and was continued within that range up to 192 hours. A significant increase in urea nitrogen level even during fluid therapy might be due to distrubance in fluid and electrolyte balance as addition of chloride ions through sodium chloride, potassium chloride and calcium chloride in the transfusion fluid did not raise the chloride level which may be due to continued sequestration of their ion in the abomasum and fore-stomach Mishra (1987). The present data indicted that it may be required to add more chloride ions in the transfusion fluid to bring to the preoperative value.

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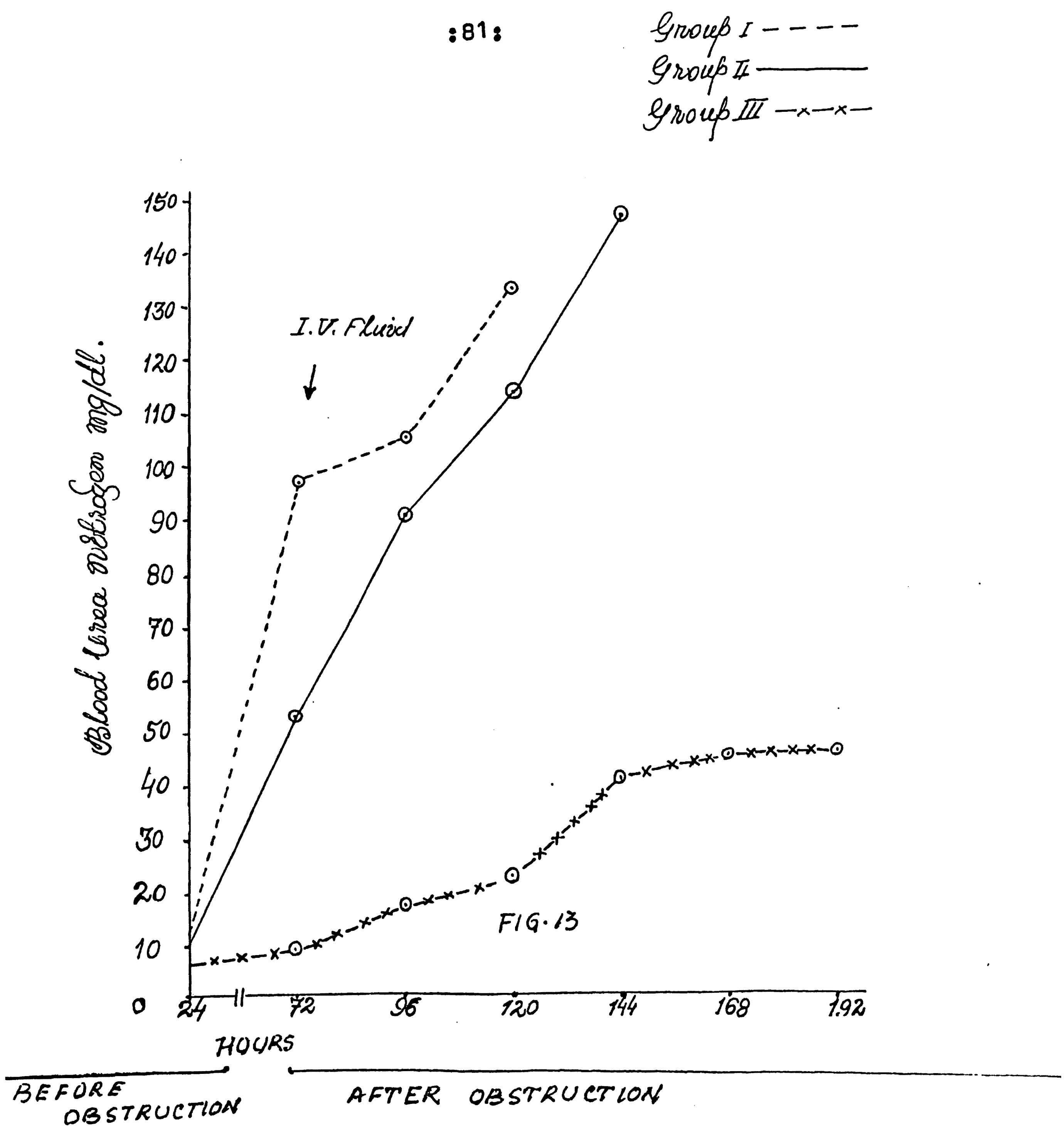
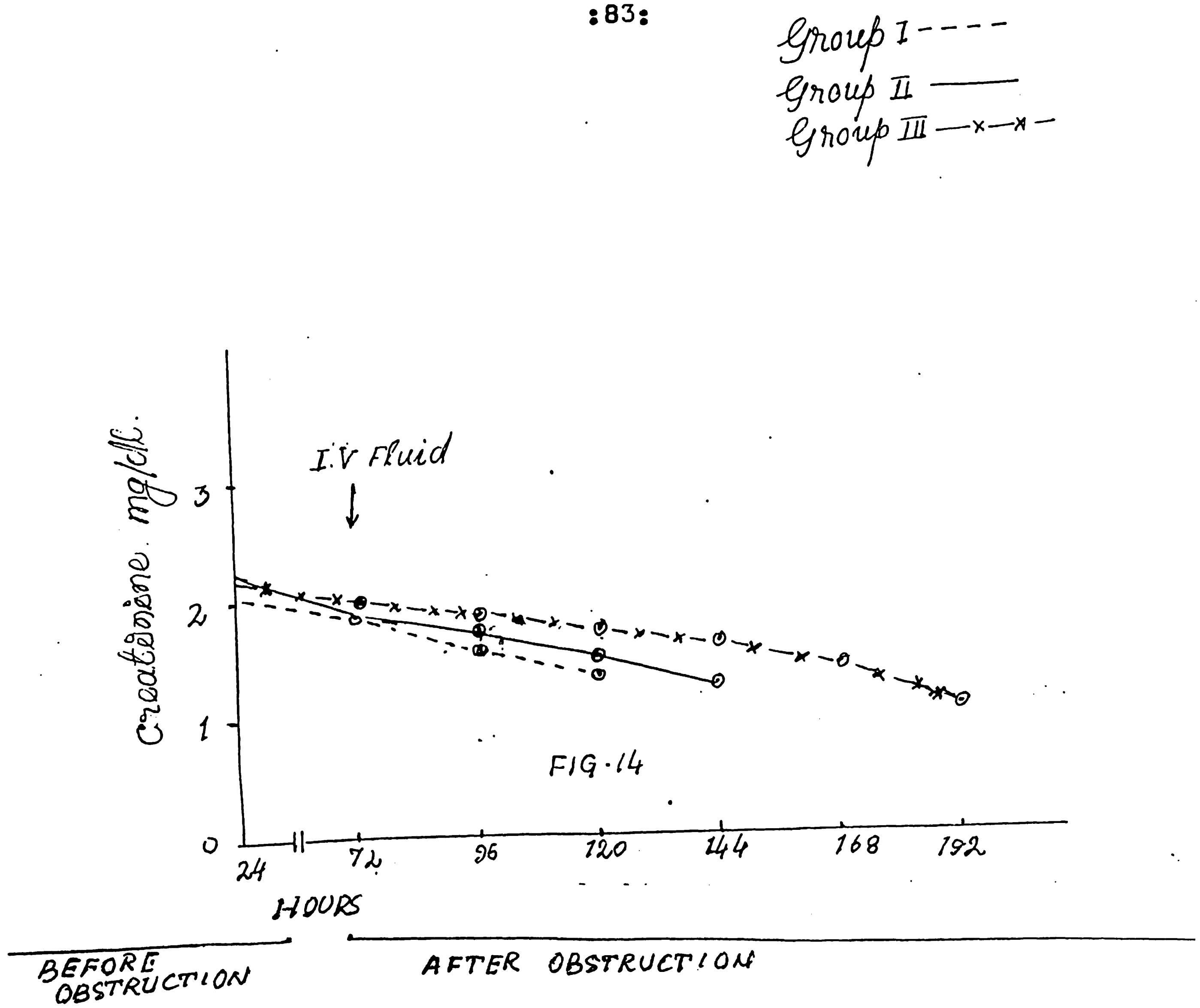


FIG. 13

GRAPH SHOWING CHANGES IN BLOOD UREA NITROGEN BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY.

evidenced by increase level of PCV indicating dehydration and distrubance in level of potassium and Chloride. Campbell (1970) has expressed similar opinion. The longivity in Group I and Group II was less in comparison to Group III might be due to abnormal elevation of urea nitrogen level in comparison to animals in Group III which survived throughout the period of experiment. This corroborates the opinion of Campbell (1970) that high blood urea level offer a poor prognosis. The continued increase in the urea nitrogen level during fluid therapy might be due to decrease hepatic circulation as a result of distended fore-stomach, impact of dehydration (Campbell,1970) and failure of recycling process of urea through the salivary gland and non utilization by the rumen microbes. Basu (1987) has stated that there was decreased salivary secretion following pyloric obstruction in his experiments. Mishra (1987) has suggested that there will be decreased rumen metabolism due to the sudden decrease in pH.

The mean serum creatinine level in all the three groups is 2.08 $\pm$ 0.05 and 2.25 $\pm$ 0.11 during the control period. It decreased significantly($P \angle 0.01$) in all the



GRAPH SHOWING CHANGES IN SERUM CREATININE BEFORE AND AFTER PYLORIC OBSTRUCTION AND AFTER FLUID THERAPY three groups (Appendix 1c, 2c, 3c) till the terminal stages. The significant decrease in serum creatinine may be due to decreased muscular activity which is the main contributor of creatinine to the circulation (Strand, 1970) and continued excretion through urine. Moreover creatinine excretion is not effected by dietary protein intake or urea levels (Yoxall and Hird, 1980). Decreased muscular activity may be due to dehydration and electrolyte imbalance as observed in present investigation. However Avery(1986) has recorded increased level of creatinine in case of induced duodenal obstruction.

* * *

S U M M A R Y

Nine cross bred Jersy calves were induced with pyloric obstructions following exteriorisation and ligation of the pylorus. They were divided into 3 groups with 3 animals in each. Clinical signs, biochemical changes in serum and whole blood and electrolytes were recorded prior to and 72 hours after ligation of the pylorus. Three different fluids having different compo-sitions were tested in each respective groups. The changes in the same animal were recorded till the animal died or 192 hours of ligation, which ever was earlier. The animal started showing symptoms of obstruction within 24 hours after ligation which was manifested by inappetance, gradual distension of the abdomen, reduced rumination, splashing sound on ballottment, decreased or cessation of defecation, reduced urination and dehydration.

Significant ($P \angle 0.01$) increase in pulse rate, heart beat significant ($P \angle 0.01$) decrease in respiration and ruminal contraction even during fluid therapy and significant ($P \angle 0.05$) fall in rectal tempeature during terminal stages were recorded.

There was significant ($P \angle 0.01$) increase in P.C.V. and Haemoglobin by 72 hours of obstruction in all the groups which decreased significantly ($P \angle 0.05$) and levelled gradually towards the normsl values during fluid therapy. Biochemical changes also indicated a significant ($P \angle 0.01$) increase in the glucose level during fluid therapy and significant ($P \angle 0.05$) fall in Potassium and chloride levels even during the period of fluid therapy. There was an increase in serum urea nitrogen, fall in creatinene level throughout the period. However, no significant changes have been recorded in serum calcium and Sodium either before or after induction of fluid therapy. The present study indicated that the fluid containing 20% dextrose with the composition of electrolytes simulating 'Rintose', a commercially available fluid has shown changes of less magnitude and increased longivity in compaison to other two fluids containing different electrolytes with 5% and 10% dextrose respectively at dose of 30 ml /kg/body weight.

The animals in group III receiving 20% dextrose with electrolytes continued to survive beyond 192 hours till the fluid therapy was discontinued. This period of time was considered to be substantially adequate to salvage the patient for a planned operation.

* * *

CHAPTER VII

CONCLUSION

C O N C L U S I O N

The present study indicated more significant changes in fluid and electrolyte balance during pyloric obstruction. Administration of fluid improved the activity and longivity of the animals. Fluid containing sodium chloride, calcium chloride, potassium chloride, sodium lactate with 20 % dextrose (Rintose) at a dose rate of 30 ml/kg body weight proved to be more effective to lengthen the longivity and to combat electrolyte imbalance in comparison to the fluids prepared by adding isotonic solutions of sodium chloride, potassium chloride, calcium chloride with 5% or 10% dextrose at the same dose rate. Hypokalaemic and hypochloraemic condition continued even during fluid therapy in all the groups and was indicative for the requirement of more potassium chloride to be added to the transfusing fluid but the quantity to be added needs further study.

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APPENDIX

Appendix 1 (a) Analysis of variance of Pulse, Heart beat, Respiration rate, Rectal Temperature and Ruminal contraction of Group I Animals.

Dependant variable	Source	DF	SS	MS	F	CD at 5%
Pulse rate	Treatment	3	2692	897.33	112.17**	4.43
	Error	8	64	8.00		
	Total	11				
Heart rate	Treatment	3	2810.35	936.75	261.67**	3.55
	Error	8	28.67	3.58		
	Total	11				
Respiration	Treatment	3	180.35	60.12	89.73**	1.53
	Error	8	5.32	0.67		
	Total	11				
Temperature	Treatment	3	7.98	2.66	6.8	1.17
	Error	8	3.13	0.39		
	Total	11				
Ruminal contraction	Treatment	8	46.92	15.64	31.28**	1.30
	Error	3	4.00	0.5		
	Total	11				

** Significant at 1% level ($P < 0.01$)

Appendix 1 (b) Analysis of variance of Packed Cell Volume, Haemoglobin, blood glucose Serum Calcium and Potassium of Group I Animals.

Dependant Variable	Source	D.F	SS.	MS.	F.	CD at 5%
Packed Cell Volume	Treatment	3	576.56	192.19	17.63**	6.21
	Error	8	87.17	10.90		
	Total	11				
Haemoglobin	Treatment	3	17.45	5.82	6.54**	1.77
	Error	8	7.10	0.89		
	Total	11				
Blood Glucose	Treatment	3	10745.38	3581.79	516.85**	4.96
	Error	8	55.44	6.93		
	Total	11				
Serum Calcium	Treatment	3	0.55	0.18	0.78	0.91
	Error	8	1.86	0.23		
	Total	11				
Serum Potassium	Treatment	3	2.04	0.68	13.6**	0.42
	Error	8	0.39	0.05		
	Total	11				

** Significant at 1% level ($P < 0.01$)

Appendix 1 (c) Analysis of Variance of Serum Sodium, Chloride, Urea nitrogen and Creatinine of Group I Animals.

Dependent Variable	Source	D.F.	SS.	MS.	F.	CD at 5%
Sodium	Treatment	3	23.59	7.86	2.26	3.52
	Error	8	27.83	3.48		
	Total	11				
Chloride	Treatment	3	1144.16	381.39	79.46**	4.10
	Error	8	38.41			
	Total	11				
Urea nitrogen	Treatment	3	24596.09	8198.70	220.80**	11.40
	Error	8	297.03	37.13		
	Total	11				
Creatinine	Treatment	3	0.86	0.29	14.50	0.26
	Error	8	0.12	0.02		
	Total	11				

** Significant at 1 % level ($P < 0.01$)

Appendix 2 (a) Analysis of Variance of Pulse, Heart beat, Respiration rate Rectal Temperature and Ruminal contraction of Group II Animals

Dependent Variable	Source	DF	SS	MS	F	CD at 5 %
Pulse	Treatment	4	2287.07	571.77	245.39**	2.77
	Error	10	23.33	2.33		
	Total	14				
Heart beat	Treatment	4	4028.93	1007.16	6.88**	21.99
	Error	10	1464	146.4		
	Total	14				
Respiration	Treatment	4	350.93	87.73	25.00**	3.34
	Error	10	34.00	3.40		
	Total	14				
Rectal Temperature	Treatment	4	9.57	2.39	3.51*	1.50
	Error	10	6.83	0.66		
	Total	14				
Ruminal contraction	Treatment	4	52.00	13.00	21.67**	1.42
	Error	10	6.00	0.60		
	Total	14				

* Significant at 5% level ($P < 0.05$), ** Significant at 1% level ($P < 0.01$)

Appendix 2 (b) Analysis of variance of Packed cell volume, Haemoglobin, Blood Glucose, Serum calcium and Serum Potassium of Group II Animal.

Dependant Variables	Source	DF	SS	MS	F	CD at 5 %
Packed cell volume	Treatment	4	61.27	15.32	4.82*	3.24
	Error	10	31.84	3.18		
	Total	14				
Haemoglobin	Treatment	4	6.51	1.63	5.43*	1.00
	Error	10	2.97	0.30		
	Total	14				
Blood Gluc-ose	Treatment	4	28280.16	7070.04	337.15**	8.32
	Error	10	209.69	20.97		
	Total	14				
Calcium	Treatment	4	0.40	0.1	0.37	0.94
	Error	10	2.68	0.27		
	Total	14				
Potassium	Treatment	4	1.60	0.04	40*	0.56
	Error	10	0.97	0.10		
	Total	14				

* Significant at 5% level (P ∠ 0.05). ** Significant at 1% level (P ∠0.01)

Appendix 2 (b) Analysis of variance of Packed cell volume, Haemoglobin, Blood Glucose, Serum calcium and Serum Potassium of Group II Animal.

Dependant Variables	Source	DF	SS	MS	F	CD at 5 %
Packed cell volume	Treatment	4	61.27	15.32	4.82*	3.24
	Error	10	31.84	3.18		
	Total	14				
Haemoglobin	Treatment	4	6.51	1.63	5.43*	1.00
	Error	10	2.97	0.30		
	Total	14				
Blood Gluc-ose	Treatment	4	28280.16	7070.04	337.15**	8.32
	Error	10	209.69	20.97		
	Total	14				
Calcium	Treatment	4	0.40	0.1	0.37	0.94
	Error	10	2.68	0.27		
	Total	14				
Potassium	Treatment	4	1.60	0.04	40*	0.56
	Error	10	0.97	0.10		
	Total	14				

* Significant at 5% level (P ∠ 0.05). ** Significant at 1% level (P ∠ 0.01)

Appendix 2 (c) Analysis of variance of serum Sodium, Chloride, Urea nitrogen and creatinine in Group II animals.

Dependant Variable	Source	DF	SS	MS	F	CD at 5 %
Sodium	Treatment	4	8.09	2.02	0.55	3.47
	Error	10	36.54	3.64		
	Total	14				
Chloride	Treatment	4	826.09	456.52	66.94**	4.70
	Error	10	68.23	6.82		
	Total	14				
Ureanitrogen	Treatment	4	18188.00	45.47	25.17**	24.45
	Error	10	1806.70	180.67		
	Total	14				
Creatinine	Treatment	4	1.69	0.42	21.00**	0.25
	Error	10	0.23			
	Total	14				

** Significant at 1 % level ($P < 0.01$)

Appendix 3 (a) Analysis of variance of Pulse, Heart beat, Respiration rate, Rectal Temperature and Rumiral contraction of Group III Animals.

Dependant Variable	Source	DF	SS	MS	F	CD at 5 %
Pulse	Treantment	6	3909.24	651.54	9.46**	14.52
	Error	14	964.00	68.86		
	Total	20				
Heart beat	Treatment	6	2996.57	499.42	36.00**	6.46
	Error	14	192.00	13.71		
	Total	20				
Respiration	Treatment	6	337.91	56.32	37.65	2.15
	Error	14	21.33	1.52		
	Total	20				
Temperature	Treatment	6	16.49	2.75	2.64	1.79
	Error	14	14.62	1.04		
	Total	20				
Ruminal contraction	Treatment	6	24.48	4.08	7.85**	1.27
	Error	14	7.33	0.52		
	Total					

** Significant at 1 % level ($P < 0.01$)

Appendix 3 (b) Analysis of variance of Packed cell Volume, Haemoglobin, Blood, Glucose, Serum calcium and Potassium in Group III Animals.

Dependant variable	Source	DF	SS	MS	F	CD at 5 %
Packed cell Volume	Treatment	6	24.79	4.13	3.97*	1.79
	Error	14	14.57	1.04		
	Total	20				
Haemoglobin	Treatment	6	2.00	0.33	0.36	1.67
	Error	14	12.75	0.91		
	Total	20				
Glucose	Treatment	6	80255.42	13375.90	9.75**	64.85
	Error	14	19202.00	1371.54		
	Total	20				
Calcium	Treatment	6	1.78	0.30	0.50	1.55
	Error	14	7.27	0.60		
	Total	20				
Potassium	Treatment	6	4.35	0.73	5.21*	0.67
	Error	14	1.95	0.14		
	Total	20				

* Significant at 5% level ($P < 0.05$), ** Significant 1 % level ($P < 0.01$)

Appendix 3 (c) Analysis of variance of Serum Sodium, Chloride, Urea nitrogen and Creatinine of Group III Animals.

Dependant variable	Source	DF	SS	MS	F	CD at 5 %
Sodium	Treatment	6	23.54	3.92	0.03	18.96
	Error	14	1641.52	117.25		
	Total	20				
Chloride	Treatment	6	1749.49	291.58	2.88*	17.59
	Error	14	1415.80	101.20		
	Total	20				
Urea nitrogen	Treatment	6	5316.06	886.01	133.63**	4.52
	Error	14	92.88			
	Total	20				
Creatinine	Treatment	6	2.53	0.42	84.00**	0.12
	Error	14	0.08	0.05		
	Total	20				

* Significant at 5 % level ($P<0.05$), ** Significant at 1 % level ($P<0.01$)